SWEAT COMPENSATED EMG AMPLIFIER USING MICROCOMPUTER 8748

A Thesis Submitted
in Partial Fulfilment of the Requirements
for the Degree of

MASTER OF TECHNOLOGY

by

LIEUT S K SINGH

to the

DEPARTMENT OF ELECTRICAL ENGINEERING

INDIAN INSTITUTE OF TECHNOLOGY KANPUR

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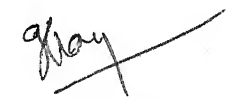
CERTIFICATE

This is to certify that the work entitled, "SWEAT COMPENSATED EMG AMPLIFIER USING MICROCOMPUTER" by Liutenant S.K. Singh has been carried out under my supervision and has not been submitted elsewhere for a degree.



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ACKNOWLEDGEMENTS

I am thankful to Dr. G.C. Ray of Electrical Engineering Department of I.I.T. Kanpur for his timely and resourceful guidance at all stages of my work.

I owe my gratitude to my colleagues who have been of great help in helping me to detect and rectify the knots at critical stages.

I also appreciate the help rendered by the Central and ACES Library in terms of invaluable data and most important of all, the ACES Stores.

I am also indebted to Mr. R.N. Srivastava for the patient and flawless typing of this thesis.

- Lt. S.K. Singh

CONTENTS

```
        OUTL P2,A;
        ENTØ CLK;          Output 2MHz clock at TØ pin.

LOOP1:  IN A,P2;           Wait for "RUN" signal.
        JBØ LOOP1;
        JB1 NORML;

TEMP:   MOV A1 # 5AH;      Voltage controlled amplifier
                           gives unity gain for a control
                           voltage of (-0.7 V), the corres-
                           ponding DAC word is 5A.
        MOV RØ, # 21H;
        MOV @RØ,A;         Save DAC word at RAM location 21.
        OUTL P1,A;         5A to DAC
        CALL HOLD;         Give delay for the circuit to
                           stabilise
        CALL ADC;          Read A/D converter, reading
                           left in accumulator.

GAIN:   MOV RØ, # 2ØH;
        MOV @RØ,A;         Reference voltage stored at RAM
                           location 2Ø.

ADJST:  CALL HOLD;         Give delay
        CALL ADC;          Read ADC
        CPC A;
        ADD A, # Ø1H;      Take 2's complement of current
                           reading.
        MOV RØ, # 2ØH;     Memory pointer (RAM) to 2Ø (i.e.
                           reference voltage).
        ADD A, @ RØ;       Add reference voltage to current
                           reading.
        JZ WAIT;           Reference voltage = current voltage.
        JNC DECR;          Reference   current, so decrease
                           the DAC word

INCR:   MOV RØ, # 21H;     Old DAC word to A
        MOV A, @ RØ;
        INC A;             Ref   current reading; so
                           increase the DAC word.
        JZ MAX;            DAC word = ØØ of incremented at FF.

NEXT:   OUTL P1,A;         New DAC word to DAC
        MOV RØ # 21H;
        MOV @ RØ,A;        Some new DAC word
        JHP ADJST;         Repeat the process

MAX:    MOV A, # ØFFH;     Set maximum DAC word and
        JMP NEXT;          Send to DAC

DECR:   MOV RØ, # 21H;     Old DAC word to A
        MOV A, @ RØ;
        JZ NEXT;           DAC word is minimum
        DEC A;
        JMP NEXT;
```

SYNOPSIS

The aim of the project undertaken was to develop on Electromyographic Channel (EMG Channel) which can pick up signals from the muscles of a person and amplify it to a level large enough to be used in pattern recognition circuits for prosthesis; or by CRO or spectrum analysers and similar circuitry used as a diagnostic tool in medical science.

EMG channel finds a very important place in medical profession. It can help analyse myopathic diseases. It is used for limb prosthesis. Demand pacemakers are another example. It has also been possible to provide, so-called, "Will operated hands" to above elbow amputee. The signal generated by twitching the shoulder muscles is picked up by an EMG channel and the voltage is then used for moving the artificial motor operated hand.

Work was started in this field in the countries abroad towards the early half of the present century. Dr. G.C. Ray, Dr. S.K. Guha, Dr. Mukhopadhyay etc. have worked in this field in India.

An EMG channel has been developed by Dr. G.C. Ray. A three electrode system was used by him. The problem faced by him was typical for a tropical country. A layer of sweat between two active electrodes poses the low input impedance at the preamplifier and the EMG channel stops picking up any

signal after some time. Dr. Ray suggested a remedial measure in which a low magnitude high frequency signal is injected into the subject. That attenuation of this high frequency voltage is taken as a measure of random set layer and this information is than used to provide AGC to the EMG channel.

The drawback of the system is open loop nature, characteristic dissimilarity between two transistors of the slope changer and the non-linear characteristic of the transistor itself.

The present work makes an effort to improve upon these drawbacks by using a single chip microcomputer. In this a reference voltage is stored in the memory and compared with the current amplitude of the high frequency signal. The computer accordingly modifies the output of a DAC which in turn controls a gain of two voltage control amplifier placed in EMG and high frequency channel respectively. As a result the loop becomes a close one. Once the current reading equals the reference a "wait period" can be introduced wherein the high frequency signal injection is cut-off and the DAC output is maintained at the previous value. A full gain adjustment cycle may take about an average of 15 to 20 seconds. The equipment will work satisfactorily with a low duty cycle of 0.2 to 5%. Since signal injection can be stopped, the equipment can be used for clinical uses also. Interaction has been made simple by using toggle switches instead of a key board.

CHAPTER 1

INTRODUCTION

Electromyography is concerned with electric phenomena occurring in the muscular tissues and its reaction to electrical stimuli. Electromyography requires the knowledge of electrophysiology of livng tissues, bio-electric phenomena associated with them and a sound knowledge of electrical instrumentation, signal processing, control systems etc. Electromyography is thus a combination of applied electrophysiology and applied electronics as well.

Historically, the presence of electricity in living organism was detected as early as in the times of Galvani and Volta around 1790. Bio-electrical science has covered miles since then and carried by great scientists like Wedensky (1883), Einthoven (1899), Erlanger (1922), Hodgkin and Huxley (1939), Schafer (1940-42) etc. to name a few.

With the advances made by electronics, it became feasible to record and analyse the electrical behaviour of living tissues. Attempts to process vast amount of data obtained by simultaneous monitoring of many electrodes, associated with the problem of detection of weak signals submerged in noise, caused the recent invasion of digital computers and the extensive use of statistical tools into the field of electrophysiology and electromyography in particular.

In the present decade much improvement has been made in this field by Dunfield, Fusfeld, Almstorm, Herberts etc.

abroad. But the advancements gifted by these men in countries abroad (especially Europe and U.S.S.R.) are somewhat limited in application in tropical countries like India. Ray, S.K. Guha and Sneh Anand are few of those who have done extensive studies in this field in India.

Electromyography has become a strong tool for dignostic usage in myopathic deseases and tissue disorders. It has been possible to fabricate artificial limbs capable of carrying out movements, by use of EMG channels. The result is the induction of engineers on the staff of electrophysiological institutions. Also it is much more important for the medical professionals to know their equipment. In this connection, the statement of Pavlov is not outdated, "Science progresses in steps depending upon the success of techniques. Each improvement in technique raises us a stage upward, from which a new horizon is uncovered containing phenomena not known before".

This thesis can broadly be divided into two halves as follows:

(1) (a) A study of electrical states e.g. polarity of tissues, cells, etc.

(b) Generation of action potential in response to a stimuli, its mode of propagation and

(c) The effect of electric currents on tissues.

(2) (a) A study of clinical usage of electromyography and

(b) Development of an electromyographic channel which is immune to the deposition of sweat layer, a common feature of tropical countries like India.

CHAPTER 2

ELECTRICAL IMPULSES IN HUMAN BODY AND ITS PROPAGATION

2.1 Introduction:

Evidences of presence of electricity in animals were obtained as late as the 18th century. The "Electric Ray" fish and the "Electric Eel" are some of the examples. Scientists like Galvani, Volta, Matteucci and Du-Bois-Raymond took these hints and went ahead to successfully develop the theory of electrical phenomena in living tissues.

By using a simple galvanometer, Matteucci showed in 1838 that the muscle exterior was electrically positive to muscle interior. He also proved that the potential difference of the state of rest declined sharply during excitation. He further proved that if a second nerve was brought in contact with a contracted muscle, it lead to the contraction of the second also. It can then easily be inferred that the potential generated due to contraction of the first muscle is strong enough to stimulate the nerve in contact.

2.2 Resting Potential -- Its Origin:

A potential difference of 60-90 millivolts exists between the outer surface of a cell and its protoplasm where the cell surface is positive with respect to the protoplasm. This potential difference is called the resting membrane potential and it can be measured.

The origin of resting potential can be explained with the help of membrane ion theory established by Hodgkin and Huxley (1952). This theory states that there exists unequal concentration of potassium, sodium and chlorine ions (the ingredients of a living cell) within the cell as compared to its exterior. Also the cell membrane offers different degree of permeability to these ions. This difference of permeability and the concentration gradient gives rise to the potential gradient.

The protoplasm of nerve and muscle cell contains 30 to 50 times more potassium ions than the extracellular fluid and 8 to 10 times lesser sodium ions and 50 times lesser chlorine ions. The plasmatic membrane is extremely thin -- of the order of 100 angstroms and this does not permit equalising of the concentration.

In the cell membrane there are minute channels or pores which are only a few angstroms in diameter. Any molecules of water and other substances and ions of different substances have to pass through these pores.

Various ions remain attached to this membrane and thus lend a particular electric charge to the walls of its pores. This accumulation of ions (hence charge) impends or facilitates the passage of other ions of same or opposite polarity respectively.

The membrane of nerve fibre is more permeable to cations than to anions. The cause has been attributed to the

presence of dissociated phosphate and carboxyl groups. This permeability of cation also varies depending upon the state of the muscle. For example, at rest the permeability of nerve fibre membrane to potassium ions is 20 to 100 times that of sodium ions whereas the ratio is reversed in case of an excited nerve fibre.

The origin of resting potential of a membrane can be very well understood by the following experiment. Take a vessel (Figure 1) and divide it into two halves by an artificial membrane whose pores allow positively charged potassium ion to pass but are impenetrable to negatively charged sulphate ions. Fill the right half of the vessel by strong K_2So_4 solution and left half with a less concentration K_2So_4 solution. Owing to the presence of a concentration gradient, the potassium ion diffuse from right half to left half. Similarly the sulphate ions also try to diffuse to left half, but are held at the right of the membrane because of their larger size. The negative charge of these sulphate ions exercise a binding force on the potassium ion on the other side of the membrane and held them on to the left half of the membrane. This is how the polarisation of the membrane occurs giving rise to a potential difference. If now a galvanometer is used it will show that the K_2So_4 solution in the left half is electrically positive to the solution on the right half.

The potential difference is given by the Nernst's formula $E = 58 \log \frac{C_1}{C_2}$ mv at 18°C where C_1 and C_2 are the concentrations of the right and left K_2So_4 solutions respectively.

A similar situation does exist in a living nerve fibre because the concentration of potassium ions in protoplasm is 30 times that in the external solution and the organic (protein etc.) anions of protoplasm do not penetrate the membrane.

In the state of rest the positively charged potassium ions from protoplasm to the extracellular fluid makes the outer surface of the membrane positive with respect to the inner surface. The potential different of 90 mv obtained across a muscle fibre proves the Nernst's formula.

Apart from the potassium ions, sodium ions diffusing into protoplasm from the extracellular fluid, also adds to the resting potential. The permeability of the membrane to sodium ions increases when the muscle is excited.

It can now be said that the resting potential of nerve fibres and cells depends upon the ratio of positively charged potassium ions diffusing out of the cell for unit time to the negatively charged sodium ions diffusing into the cell. The higher the ratio greater the resting potential.

2.3 Action Potential:

All cells can pass from a state of physiological rest to one of excitation in response to a stimuli. In general the nerve and muscle tissues, which give rise to an electrical impulse, travelling along the membrane, in response to a stimulus, are called excitable tissues.

Excitation may be characterised by an aggregate of electrical, temperature, chemical, functional and structural changes in the living cell of which the bio-electrical phenomena are the most important.

If an electric current (of sufficient magnitude and duration) is applied to a living tissue, it gets excited and it shows in the form of rapid variation of the membrane potential. This potential is called the action potential.

Figure 2 shows a sample of action potential. The resting potential was 85 mv. When the external stimuli was applied the potential began to fall sharply, became zero and rose to about 30 mv in the opposite direction indicating a change in the inner surface of the membrane from being electrically negative to electrically positive with respect to the outer surface. Then a restorative process started bringing the membrane potential to the original state.

Since the initial polarization of the membrane vanishes in the ascending phase of the action-potential, it is called the depolarization phase and the descending phase is called the repolarization phase. The length of an action-potential may vary from 0.1 to 5 msecs.

2.4 After Potential:

The action potential is always followed by an after potential which may be positive or negative. These are of few millivolt amplitude and may be a few milliseconds to hundred milliseconds long. The after potential is an outcome

of the restorative process of the tissue.

Figure 3 shows the action potential of a squid axon which shows positive after potential.

2.5 Origin of Action Potential:

It has been stated earlier that a potential difference develops due to the presence of a concentration gradient of ions across a semipermeable membrane. Action potential is generated because of the change in permeability of the membrane due to the stimuli applied.

At rest, the cell membrane is more permeable to potassium than to sodium. As a result the positively charged potassium ions flow from the protoplasm to the extracellular fluid exceeds the reverse flow of sodium cations. Therefore, at rest, the outer side of the membrane is electrically positive to the inner one.

When a stimulus is applied the membrane permeability increases for the sodium ions and becomes ten times that of potassium ions. As a result the flow of sodium ions into the cell exceeds the outflow of potassium ions out of the cell, thus reversing the sign of membrane potential. Now the outer surface becomes positive with respect to the inner surface. This is the depolarisation phase.

However, the increased permeability to sodium ions is short lived and a restorative process begins soon. The sodium permeability of the membrane now falls. This is called inactivation. Repolarisation takes place.

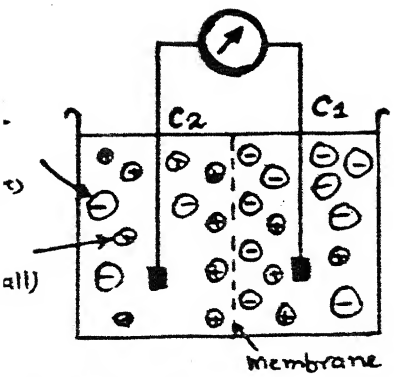
After potentials are also explained in the light of the above mentioned change in permeability. The membrane permeability to potassium ions may remain increased, even after the termination of action potential, as compared to the initial level, thus giving rise to positive after potential. Also an increase in the flow of potassium ions from the protoplasm leads to an increase in the membrane potential i.e. after hyperpolarisation.

The reverse is true for negative after potential.

2.6 Propagation of Action-Potential:

Action-potential arising in an excited cell becomes a stimuli for the adjoining cells and hence sympathetic excitation starts in the adjoining cells also. Since the magnitude of action potential (110 to 120 mv) is too large compared to the threshold of stimulation for a tissue, an undamped wave of excitation travels all along the excitable tissue.

The mechanism of conduction of excitation from one portion of the fibre to the other portion is same as the generation of action potential. Refer to Figure 4. In both the cases when the depolarisation reaches a critical value (the stimulation threshold), an action potential is generated. When current is injected into a part of the excitable tissue, the depolarisation begins at the cathode (will be explained subsequently), an electric current flows between the excited (electronegative) and resting (electropositive) parts of the membrane. The speed of conduction may be as high as 120 metres per second.



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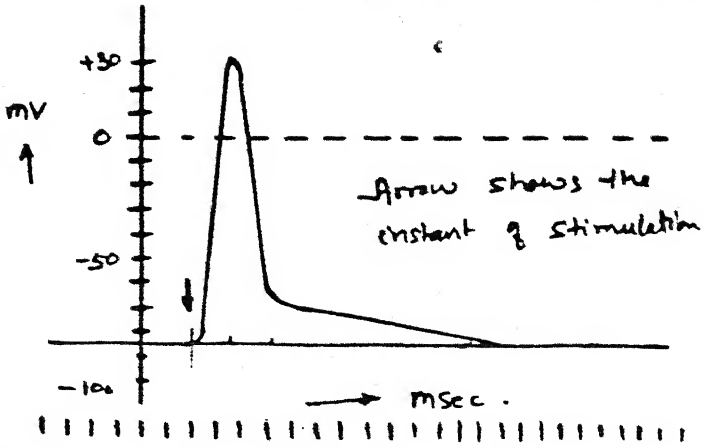
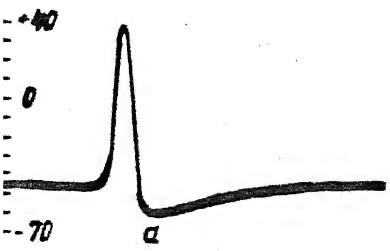


Fig. 2: Action potential of skeletal muscle registered by intracellular microel



A squid axon showing
positive after potential

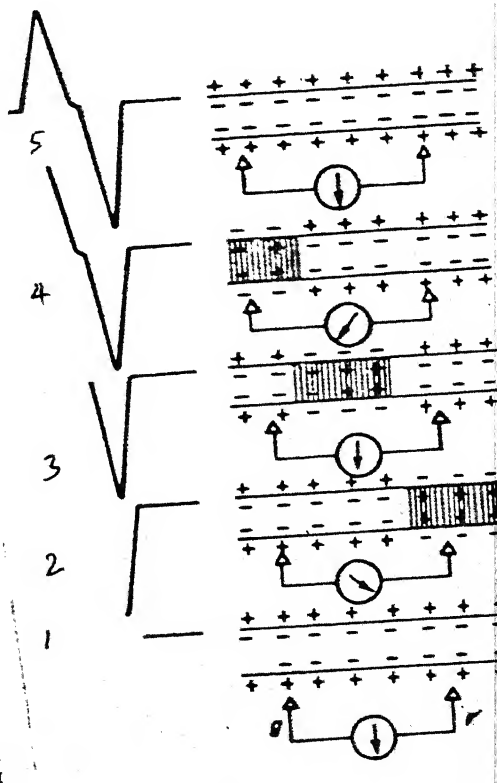


Fig. 4: Mechanism of diphosic potential generator

CHAPTER 3

THE EFFECTS OF CURRENTS ON LIVING TISSUES

3.1 The Laws of Stimulation

Any agent which can sharply increase the membrane permeability to sodium can excite a tissue. Nerve and muscle fibres can be excited by electric currents or mechanical actions like pinching, acids, alkalies, temperature gradient etc.

Out of all these stimuli, the electric current is important because

(a) it can be easily dosed in strength, duration and gradient,

(b) it does not damage a living tissue (if the strength is below a critical value) and its action is quickly reversible.

For a stimulus to cause excitation it must have necessary strength, must be applied for sufficient duration and must be steep enough. A few terms related to the above parameters are explained in the following paragraphs.

The lowest strength of stimulation which can give rise to an action potential is called the threshold of stimulation. A stimulus below it is called subthreshold and above it is called suprathreshold. Obviously for electric currents, the threshold must be expressed in units of current or voltage. The threshold of stimulation is liable to change depending upon physiological condition of the tissue and the method of stimulation.

An electric current may be applied with the help of extracellular or intracellular electrodes, the latter being more accurate because it prevents branching of the current.

The minimum duration for which an electric current must be applied, to a tissue to cause an excitation, is inversely proportional to its voltage and strength. Figure 5 shows a strength-duration curve for the exciting current. From the graph it is seen that a current below a minimum strength or voltage does not cause excitation irrespective of the duration of application. This is called the rheobase. The minimum time for which a current equal to rheobase must be applied, to cause excitation, is called the utilisation time which implies that further prolongation of the current is fruitless. If the current is increased, the time of application reduces. However there is a minimum time for which a current must be applied (irrespective of how large is the amplitude) to cause excitation. It has been observed that the rheobase undergoes a continuous slight variation depending upon different physiological states of rest. Therefore a new term chronaxie was defined. It is the least time for which a current of double the rheobase must be applied to cause excitation.

The threshold of stimulation depends on the duration of stimulus and the steepness of rise as well. If the excitation is induced by a rectangular pulse (the steepness of change is maximum), the threshold of stimulation is minimum. The threshold of stimulation increases inversely with respect

to decrease in steepness. Also, if the steepness of rise is less than a minimum, no action potential shall develop, no matter how great is the final current strength. This happens because, if the rate of increase is slow, active changes take place inside the tissue for sufficient time so as to raise the threshold of stimulation. This phenomenon of adaptation of excitable tissue to a slowly increasing stimulus is known as accommodation (Figure 6). The higher the accommodation, steeper must be rise in current. Accommodation happens in case of thermal or mechanical stimuli also. Because of accommodation only quick tapping of a nerve with a rod causes excitation whereas the same nerve pressed slowly with the same rod does not produce an excitation.

3.2 Effect of Direct Current on Living Tissue:

A direct current polarises the tissue. A phenomena peculiar to a living tissue is that on application of direct current, the excitation arises at the cathode when the circuit is made and at the anode when the circuit is broken. Also the threshold of excitation at the moment of opening the current circuit (i.e. excitation originating at the anode) is considerably higher than its closing (excitation originating at the cathode).

Let us try to understand the above phenomena. It is obvious that the passage of electric current through a living tissue, produces changes in the membrane charge. The region where the anode is placed, the positive charge on the outside

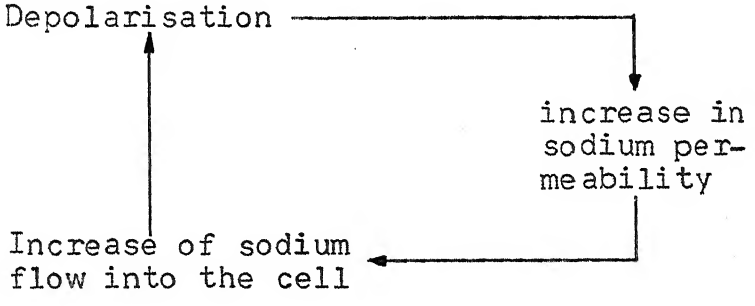
of the membrane increases causing hyperpolarisation and vice-versa. Figure 6 shows the behaviour of the membrane potential. In both the cases of increase and decrease of current, the excitation builds up and then ceases exponentially. This exponential drop is due to the capacitive behaviour (Figure 7) of a surface membrane. The outer and inner surfaces of the membrane form the plates and the layer of lipoids form the dielectric of fantastic strength (120 mv across a few angstroms). The pores in the membrane which allows ions to flow, makes it a leaky capacitor.

The rate of change of membrane potential depends upon the resistance R and capacitance C or the membrane time constant.

Changes in membrane potential occur at certain distances from the poles also, though of diminishing amplitude. These potential changes are called <u>electrotonic</u> changes and may be <u>catelectrotonic</u> or <u>anelectrotonic</u>. These are termed passive changes because of their purely physical nature.

A rise in membrane potential at the anode (passive hyperpolarisation) is not accompanied by change in ion permeability even when a strong current is applied. This is the reason why no excitation occurs at the anode when a d.c. circuit is closed. On the other hand a fall in membrane potential near the cathode (passive depolarisation) first causes a brief increase in permeability to sodium ions and then a slow increase in permeability to potassium ions (Figure 8).

The sodium permeability starts rising when the current reaches 50 to 80% of the threshold and rises further till action potential appears. The increase in sodium permeability does not peak immediately. First of all, the depolarisation of the membrane at the cathode causes a relatively slight increase in sodium permeability. Then, as the positively charged sodium ions begin to enter the protoplasm, depolarisation of the membrane increases, leading to consequent considerable rise in sodium permeability and hence further depolarisation which again increases sodium permeability. This is called <u>regenerative depolarisation</u>.



It is believed that the pores through which sodium ions can diffuse into the cell are plugged in a state of rest by calcium ions (being larger in size) and these calcium ions pass out of the pores when the depolarisation occurs in response to a stimulus and thus makes way for the sodium ions.

The sodium permeability remains increased only for 1/10th of a millisecond and then it starts reducing. This cannot be raised again by active depolarisation due to the property of inactivation. This inactivation of sodium permeability ultimately leads to triggering the repolarisation

3.3 Critical Level of Depolarisation:

It can now be understood that the depolarisation of a membrane below a critical level leads to generation of action potential and it is a property of the membrane alone (Figure 9). With a weak current the depolarisation is slow and vice-versa.

3.4 All or None Law:

This law states that a subthreshold stimulation produces no excitation while threshold stimuli produces maximum excitation immediately and is unaffected by a further increase in stimulus strength. This merely states that either no single muscle muscle fibre is excited or all of them get excited together.

3.5 Lability:

We have so far been discussing the origin and propagation of excitation through a single nerve fibre or muscle fibre. However in a living organism, rhythmic discharge of action potential, rather than isolated, pass along nerve fibres. In the sensory nerve endings e.g. receptors in skin, muscles etc., rhythmic discharge of impulses occur and travel along the fibres even with brief stimulation.

The discharge frequency of impulses vary depending upon factors like strength of stimulus, physiological conditions of the tissue etc.

Now the question arises as to what should be the minimum interval between the impulses in a rhythmic series.

It has been seen that if this period is equal to the absolute refractory period, only two impulses can be launched but third fifth etc. fail to evoke a response. Therefore to reproduce the rhythm of stimulation in a series of stimuli, the interval between them should be more than the absolute refractory period. For example the refractory period is warm blood animals is about 0.5 milliseconds whereas the maximum rate of stimulation is about 1000 pulses per second.

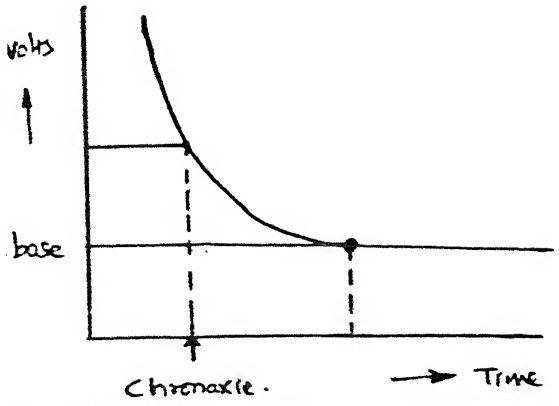
The lability can be defined as the largest number of action-potentials, an excitable tissue is capable of generating per second in response to recurrent stimuli. Lability of motor units is more than that of muscle fibres. Further, lability may vary during the course of stimulation.

3.6 The Sodium-Potassium Pump:

It has now been clearly understood that if a tissue is subjected to an impulse the number of sodium ions in the protoplasm is increased whereas that of potassium ions is reduced. Further, if a series of impulses is applied the change in number of the different ions may be significant to cause a change in the concentration gradient leading to leveling out of the inherent difference in the ion concentrations. But a special phenomena associated with the membrane actively removes sodium ions from the p otoplasm and feeds it with potassium ions. This mechanism is known as sodium-potassium pump. To do this certain amount of work has to be done and the energy is derived by metabolism. The energy required is less at rest for obvious reasons.

The power required from the pump is derived from the ATP who are high energy phosphorus compounds. The breakdown one one gram molecule of ATP generates 8000 to 10000 calories of energy. This breakdown is effected by the enzymes who are sensitive to sodium and potassium ions.

It is believed that there are some carrier agents who bind the ions and then carry them in combined form to the other side of the membrane where the ions are shed into the fluid.



ig. 5: Strength-duration curve

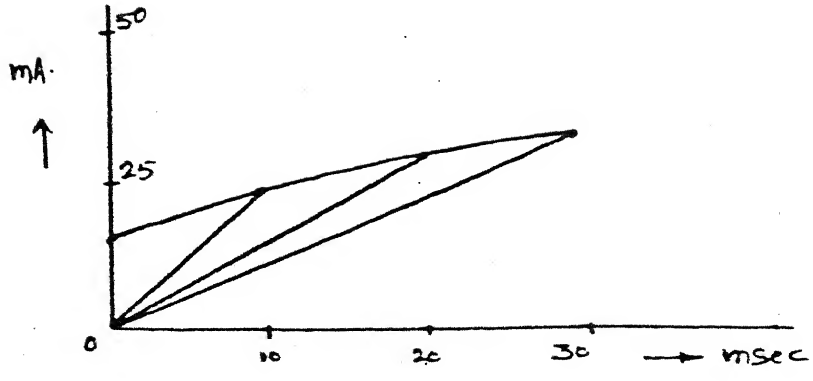


Fig. 6: Accomodation of nerve fibre

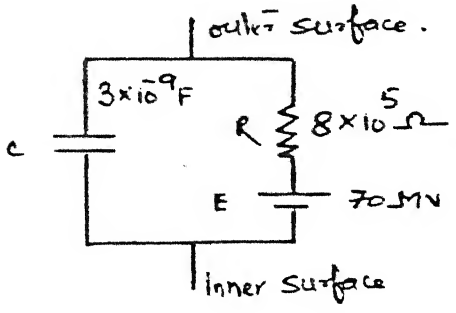


Fig. 7: Electrical Properties of a membrane.

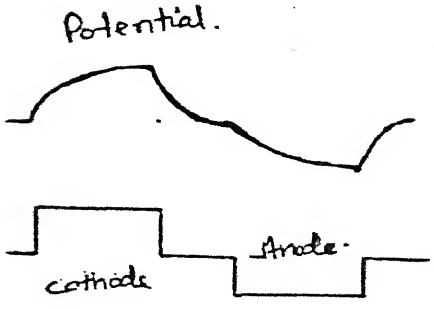


Fig. 7a: Depolarization of a membrane.

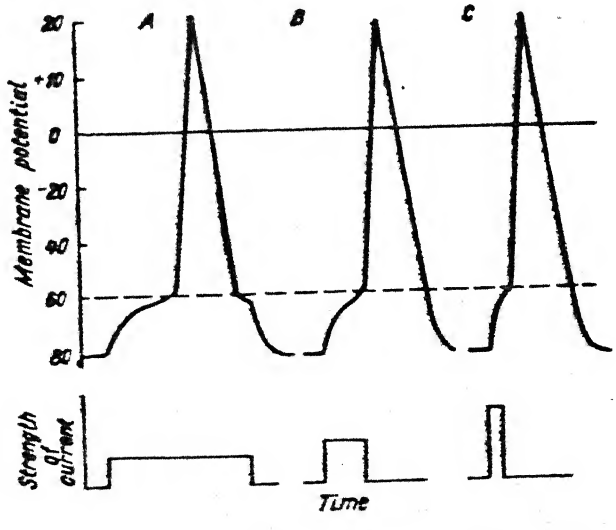


Fig. 9: Changes in membrane potential at the cathode of a stimuli

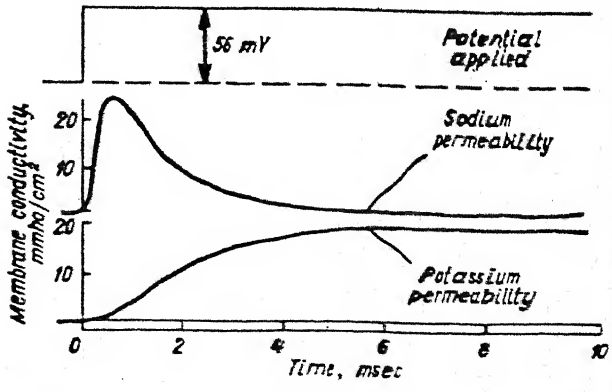


Fig. 8: Changes in sodium and potass permeability of a membrane during depolarisation by a direct current

CHAPTER 4

ELECTRICAL PROPERTIES OF MUSCLES

4.1 Introduction:

Vertebrates have three types of muscles e.g. smooth (unstriated) which form the walls of the hollow organs; striped (striated) muscle of the heart and striped skeletal muscles.

The skeletal muscles contain fibres of varying lengths and the diameter of these fibres may vary from 10 to 100 microns. These fibres are symplastic and multinuclear where the separation between cells is not defined. The fibre is enveloped in a transparent sheath called the Sarcolema. The fibres themselves are made up of sarcoplasm which contain myofibrils and sarcosomes and mitochondria.

4.2 Contraction of Muscles:

The principal physiological properties of the muscles are excitability, conductivity and contractivity. The muscles always contract in response to a stimuli.

If during the contraction of a muscle, the fibres get shortened but tension remains constant, it is called isotonic contraction. The example is a muscle lifting a load. On the other hand if the length of the muscle remains fixed (in case it is fixed at both ends), the resulting contraction is called isometric contraction.

The muscle can be excited directly by application of a current, direct or indirect when excited by a motor nerve.

The action potential generated as a response to the stimuli may change from 80-90 mv at rest to 120-135 mv when excited fully. The duration may be 3 to 5 milliseconds and the lability is 200 to 250 pulses per second. The rate of conduction of excitation in muscle fibres of biceps of man are 3.5 to 5.0 meters per second.

When a muscle is subjected to rhythmic stimulation, the contraction is intense and continuous and this phenomena is called tetanus. This is just a result of summation of individual contractions. The tetanus may be complete or incomplete depending upon whether the frequency of impulses is low or high respectively.

The contraction is greatly controlled by the motor units. Each motor unit consist of a large number of nerve fibres which contain motor cells. The number of motor nerves in a muscle may vary from 10 to 3000. Even if the muscle fibres of each motor unit are excited synchronously, the fibres of different motor units of the same muscles respond asynchronously which lends smooth movement to actions.

If rhythmic stimuli are continued for a long time, the amplitude of contraction of the muscle goes on reducing and vanishes finally. This leads to temporary reduction of the working capacity of the muscle and is called fatigue.

CHAPTER 5

THE CENTRAL NERVOUS SYSTEM AS A ELECTRICAL CONTROL ROOM

5.1 Introduction:

The central nervous system receives the impulses arising through the stimulation of the receptors in the organs and tissues, analyses them and sends back a series of impulses so as to induce or suppress the activity of peripheral organs. The accuracy of this control channel is mainly due to the formation of a closed loop between the nerve centres and the peripheral organs. Any activity induced by the impulses sent by the nerve centre is accompanied with the appearance of impulses in the receptors of the peripheral organs, thus providing a feedback.

5.2 Element of Nervous System:

The main element of the nervous system is the neurone whose job is to receive the stimulation, generate the nerve impulse and transmit them to other cells. Figure 10 shows a neurone. The neurones may vary from 6 to 180 microns in different organisms. Each neurone consists of a soma or body and processes divided into axons and dendrites. The axon conveys the nerve impulse from the nerve cell to other cells or peripheral organs. Dendrites are numerous processes emanating from one nerve cell whose job is to receive impulses arriving from other neurones and convey them to the soma. The body and processes of a nerve cell are covered with a membrane

which is permeable to potassium ions at rest and to sodium ions when excited. The resting potential is 70 mv and action-potential is 110 mv, which may extend for a duration of 1 to 3 millisecond. The level of critical depolarisation is 20 to 35 millivolts.

It has been established that protoplasmic continuity between the processes of the nerve cell exist only in lower invertebrates. In higher invertebrates and vertebrates, the nerve cells of the central nervous system are connected with one another only through synapses. Figure 11 shows that the axon divides into numerous endings into the body and dendrites of the adjacent cell. For example one nerve fibre can form 10000 synapses in other cells. The synapses located on the soma are called axosomatic and on dendrites are called axodendritic. The mediator produced in the nerve endings of these synapses is freed from its bound state through the action of an incoming nerve impulse and is secreted into the synaptic cleft. This mediator than interacts with protein-lipoid compounds resulting in the increase of sodium ions and finally depolarisation appears giving rise to an action-potential. The action-potential is first generated in the initial segment of the axon, since it has the lowest level of depolarisation.

5.3 Nerve Centre:

A group of neurones acting together, in the performance of a reflex or in the regulation of a particular function, is called a nerve centre. These nerve centres are capable of

changing the rhythm of ingoing impulses. Therefore the frequency of the impulses sent from the central nervous system to the effector organs is relatively independent of the rate of stimulation. This is due to the length of the exciting postsynaptic potential which triggers a second, third etc. action-potential.

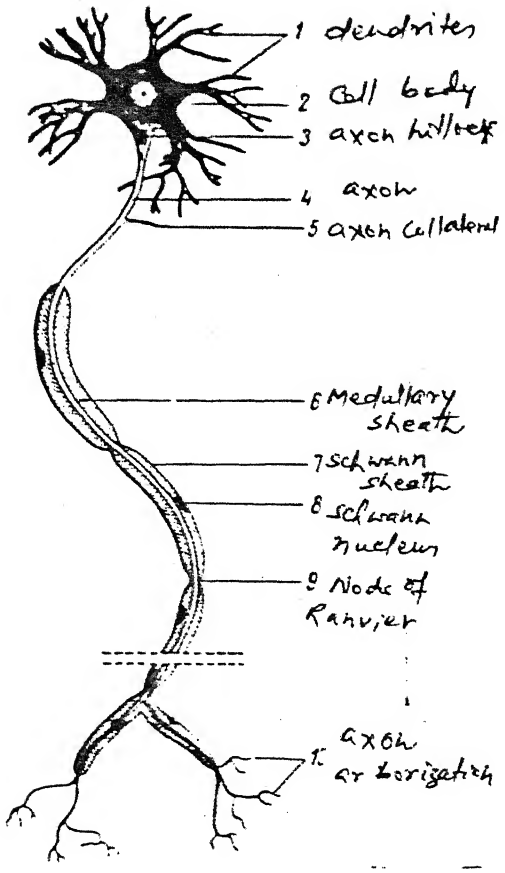


Fig. 10: A neurone

Fig. 11: Arrangement of synapses on the neuron body and dendrites

CHAPTER 6

ELECTROMYOGRAPHY - GENERAL ASPECTS

6.1 Introduction:

The science of recording electrical phenomena which accompanies muscular activity is known as electromyography. This can be recorded using surface electrodes or using microelectrodes which can be injected into the muscle.

6.2 Spectrum of EMG Signal:

In order to be able to extract the EMG signals which are deeply immersed in noise and are relatively weak signals; it is required to do some sort of S/M ratio enhancement. A crude method would be to gain the knowledge of EMG signal spectrum and then do selective filtering [2].

We have seen in the earlier chapters that the central nervous systems controls the muscular movements. There are different sets of neurones located in different places of the nervous system. When these nerve cells are excited they send a series of impulses to the so-called motor units. The rate at which these impulses are sent, to the motor units, is called the firing frequency of the motor units and this depends upon the refractory period of the tissue.

Surface EMG signals consist of two major components [3]. The first component is the firing frequency of the motor units. It has been seen that the spectral peaks contributed by these

frequencies, in the frequency domain, depend on the firing statistics of the individual motor units. The mean frequency of firing of the motor units is around 40 Hz and a dominant peak can be observed at this frequency. The motor unit firing frequency harmonious are also present in the spectrum.

The second component is the resulting frequency spectrum of the motor units action potential.

This component is heavily influenced by the recording technique, type of electrodes, distance between the electrodes, fatigue etc. The power of the EMG signal is actually compressed towards the lower frequency end for a fatigued muscle. The spectral content of a single myoelectric fibre goes up into the KHz region. The recording of such a signal can be done only by a microelectrode. If surface recording technique is adopted, the surface EMG recorded extends only upto 500 Hz. This happens because of the filtering effect rendered by the recording arrangement. The surface EMG is basically the superposition of many motor units potential and in the process the shape of individual action potential may be lost.

Figure 12 shows the spectrum of EMG signal obtained by FFT and using a Hamming Window. The graph is plotted on a linear scale. The general spectral envelope can be seen in Figure 13 which has been plotted on a logarithmic scale.

It can very well be seen that EMG spectrum has many peaks in 10 to 200 Hz region and it contains majority of the information. The higher frequency region contains almost negligible power and hence information.

6.3 Recruitment and Its Effect on the EMG Spectrum:

It has been stated that all the motor unit fibres do not fire together in response to a stimuli. In fact the number of motor units which come into action at any time, is decided by force of contraction to be exerted by the muscle.

A muscle contain a large number of fibres. These fibres are tied into bundles of varying sizes and are known as motor units which run parallel to each other from end to end [4]. Depending upon the increase in force requirement more number of motor units become active. That various motor units differ in size, can be understood by observing the smooth movement of limbs etc. even for varying degree of load. This happens because a large motor unit fixes only if all smaller motor units have fired. The total time for all fibres in the motor unit to contract, is comparable to the sum of individual motor unit activation time.

This phenomena of gradual firing of the motor units of a muscle is called recruitment. When a MU is first recruited it fires at a fixed frequency. For the bicep of a man it is 7 impulses/sec. A MU which is active increases its firing frequency by 0.01 impulses per second [4] for every grain increase in the total muscle force. Figure 14 shows the relation between number of motor units recruited for different levels of total muscle force. Figure 15 shows the variation in EMG signal power spectrum in different bands depending upon total muscle force and Figure 16 shows the

percentage of total EMG power in specified frequency band as a function of total muscle force.

6.4 Relation Between Surface EMG and Muscular Force:

The previous para showed that the amplitude of EMG signal should increase with the increase in muscular force. What is the law governing this increase? It has been found to be linear [5].

When the force level increases, motor units of gradually increasing size come into operation. Also the rate of discharge of motor units, already active, is increased. This is called rate coding.

If the rate coding is neglected, all the motor units of a particular muscle will fire at the same frequency at all force level. This will lead to a variation in the surface EMG proportional to the square root of muscular force. But rate coding makes it linear.

A freshly recruited MU will fire at 7 Hz with an incremental rate of 1.4 Hz/N. If for example we take a force level of 20 N, the firing frequency of this motor unit will become (7 + 1.4 x 20) or 35 Hz. This will give rise to a 35 Hz component in the EMG signal. In case of overlap between the action-potential developed by other motor units, the spikes may get smoothened.

As shown by Dr. G.C. Ray and Dr. S.K. Guha 6 we find that the surface EMG signal is also effected by the velocity of muscular contraction. The relationship has been shown graphically in Figure 17.

6.5 Electrodes for Surface EMG:

Different types of electrodes are used in electrophysiology, depending upon the requirements for accuracy, reproducibility of results etc. Generally metal electrodes like wires and plates of silver, platinum, nickel etc. are used. These electrodes have low resistances. But it is not recommended to use them for long stimulations. The reason is that ions from the electrodes pass into the tissue under the action of current and may have a toxic effect. Also while recording D.C. potential, there is a chance of polarisation of these electrodes due to formation of electrochemical cells.

There are non-polarisable electrodes like calomel electrodes or silver chloride electrodes. Calomel electrodes are made of mercury covered by a layer of calomel i.e. (Hg_2Cl_2) suspended in 0.9% KCl or NaCl. When this electrode is used as anode, anions from the tissue accumulate on it, especially Cl^- and form Hg_2Cl_2 which does not change the characteristic of the electrode. When this is used on a cathode, tissue cation react with Cl^- and produce Hg, again leaving the electrode composition unaltered.

Use of surface electrodes for recording EMG signals is called non-invasive technique. If micro-electrodes are used for EMG recording we call it invasive method. The micro-electrodes are capillary tubes drawn from glass.

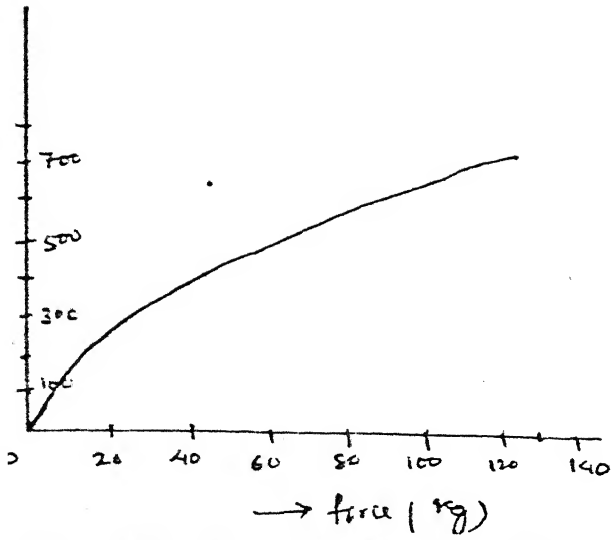
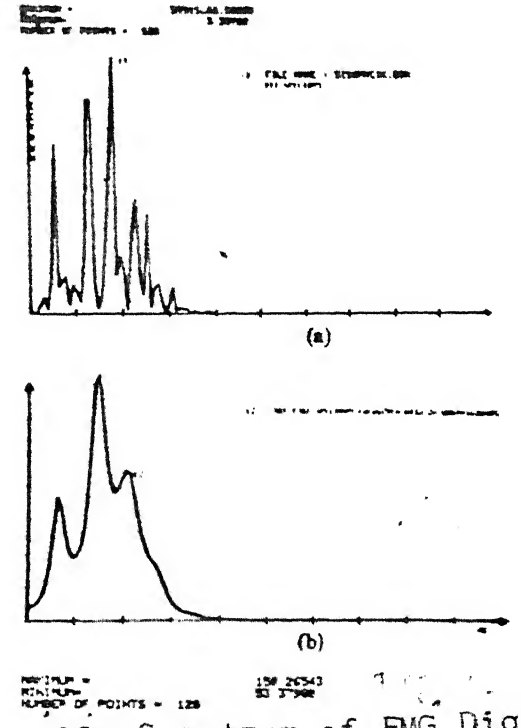
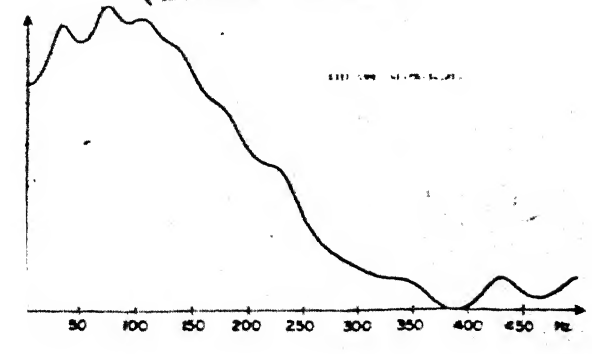


Fig. 14: No. of M4 recruited for different level of total muscle force.



. 12: Spectrum of EMG Digital (linear scale)



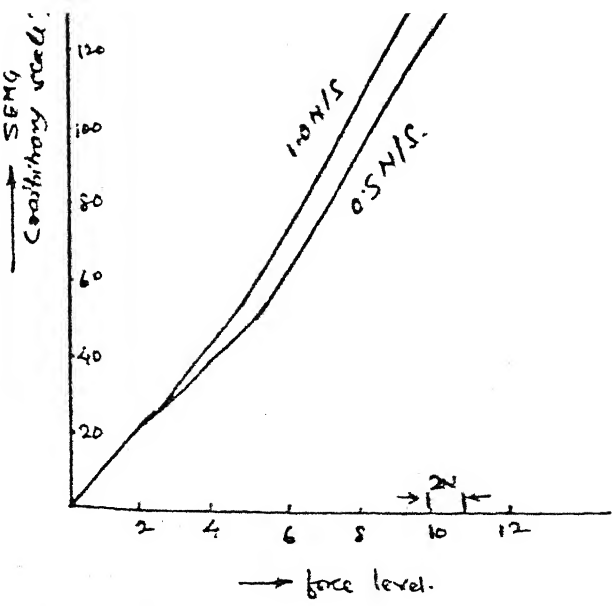


Fig. 17: SEMG variation w.r. to force level at two contraction velocities.

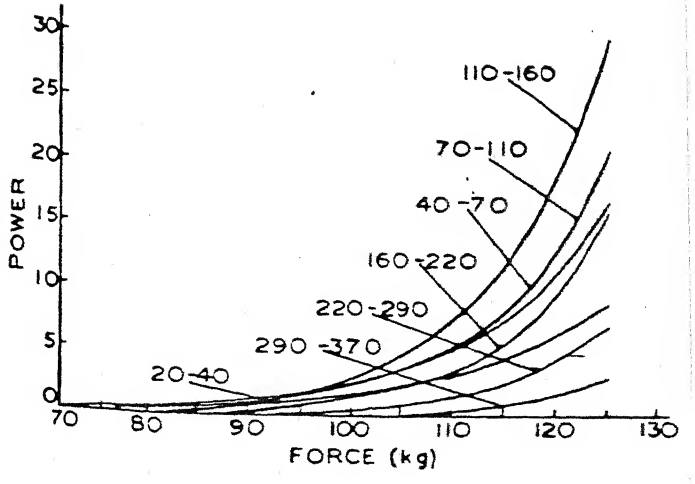
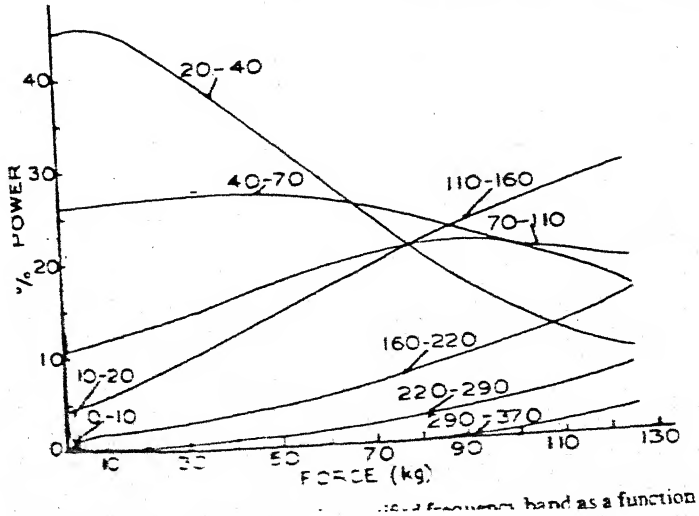


Fig. 15: EMG signal power in different ba



...ed frequency band as a function

CHAPTER 7

A TYPICAL EMG CHANNEL

7.1 Introduction:

After having studied in detail the method of generation of myoelectric signal, its mode of propagation and the spectral spread, we can now go forward for designing an EMG channel.

Figure 18 shows the block diagram of the EMG channel. The EMG potentials were picked up using silver electrodes spaced 2.5 cms apart. A three electrode system is advised to take care of the floating earth. The earth electrode was placed 3 cms away from the active electrodes. The electrodes were held in position by rubber bands.

The EMG signals picked up are first passed through a buffer to provide minimum loading of the signal and thus retain its shape. The differential amplifier was designed to give a common mode potential as low as 20 mv using LM324 quad op-amp. This CMRR is good enough for picking up the EMG signals. The signal was then band pass filtered in a band of 110 to 280 Hz (as most of the EMG signal power lies in this band). This is a crude method of increasing the signal to noise ratio for a signal of known spectrum. Since the EMG signal amplitude also depends upon the individual, the muscle selected and other physiological factors, a variable gain stage was provided to obtain suitable level at the output of

the channel. The need for a full wave rectifier stage is evident in order to obtain a d.c. output. Before finally taking out the channel output a low pass filter stage has been added to reduce the ripple content of the signal.

Figure 19 gives the circuit diagram of the EMG channel used. There is hardly anything to explain as far as the buffer and differential amplifier (gain 10) are concerned.

The design considerations for the band-pass filter are given below.

7.2 Design of Multiple Feedback Band Pass Filter:

A multiple feedback design has been preferred over VCVS design because of better cut-off characteristic obtained by the former.

Figure 20 shows a basic band pass (multiple feedback) circuit. The voltage transfer function is

$$\frac{V_o(s)}{V_1(s)} = \frac{-s(1/R_1 C_4)}{s^2 + s(1/R_5)(1/C_3 + 1/C_4) + (1/R_5 C_3C_4)(1/R_1 + 1/R_2)}$$

$$H_o = \text{Band pass gain} = \frac{1}{(R_1/R_5)(1 + C_4/C_3)}$$

$$W_o = \text{Centre frequency} = \left[(1/R_5 C_3C_4)(1/R_1 + 1/R_2)\right]^{1/2}$$

$$1/\Theta = = \left[1/R_5(1/R_1 + 1/R_2)\right]^{1/2} \left(\sqrt{(C_3/C_4)} + \sqrt{(C_4/C_3)}\right)$$

Based upon the above formula the different components were calculated to be R_1 = 6.8 K, R_2 = 27 K, R_5 = 330 K, C_3 = 0.22 Mfd and C_4 = 470 Pf.

7.3 The Full Wave Rectifier:

The requirement is to rectify the EMG signal (band pass filtered) as accurately as possible. A precision full wave rectifier has been made using a precision limiter circuit.

Figure 21 shows the precision limiter. When $e_3 > 0$ (i.e. $e_1 < 0$), i_3 is zero because $e_2 = 0$ (virtual ground) and hence D_2 is back biased. The whole of i_1 passes through the feedback resistor R_f. Then

$$e_o = -\frac{R_f}{R_1} e_1 \qquad \text{for} \qquad e_1 \quad 0.$$

The use of opamp in the circuit reduces the rounding effect of the diodes at low current.

When $e_3 < 0$ (i.e. $e_1 > 0$), diode D_1 is back biased and now all the input current flows through D_2. This leads to the output voltage to be very small, zero for all practical purposes. The limiter characteristic is also shown.

Figure 22 shows the realisation of a full wave rectifier using a precision limiter. The low pass filter is for smoothing the ripples.

In the circuit realisation for the EMG channel the filter and the inverter stage of the rectifier has been combined together.

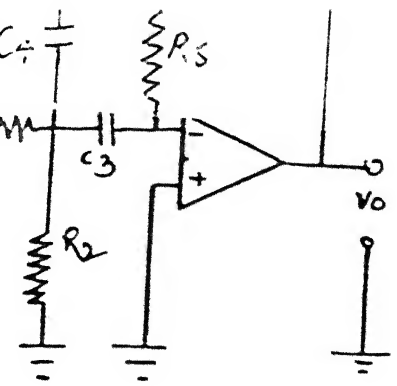


Fig. 20: Multiple feedback band pass filter

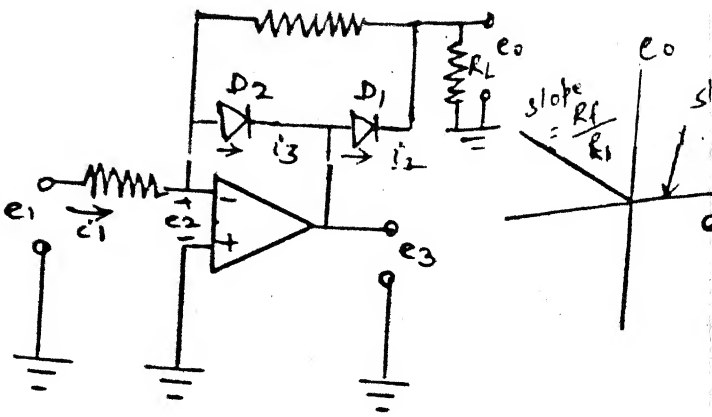


Fig. 21: Precision Limiter and its characteristics.

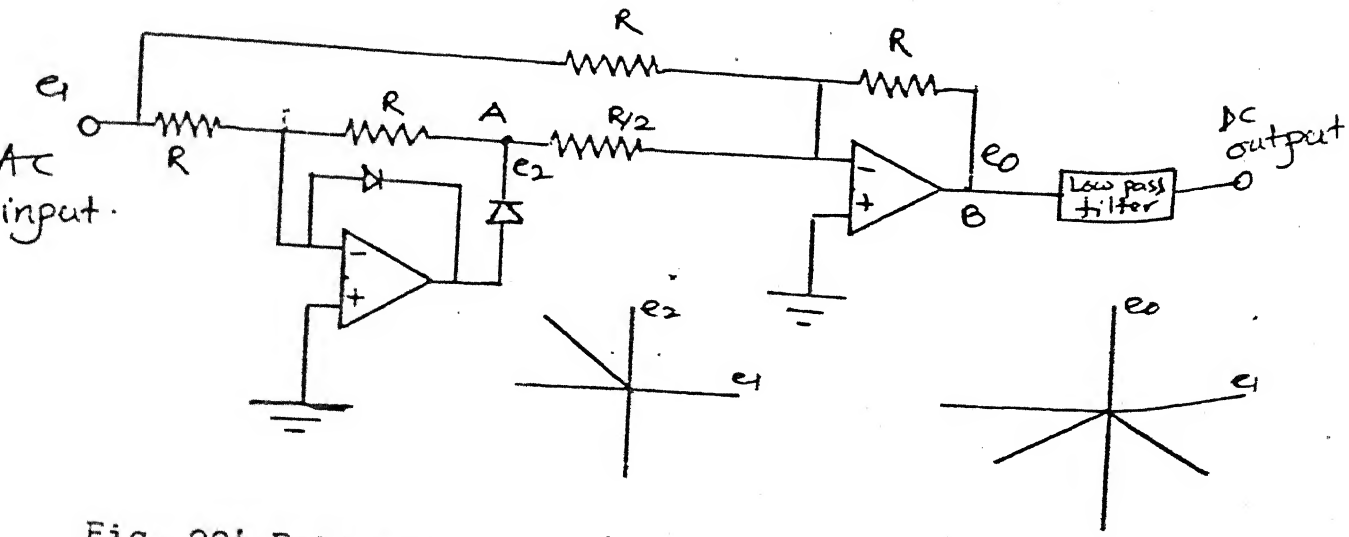


Fig. 22: Full wave rectifier and its characteristic

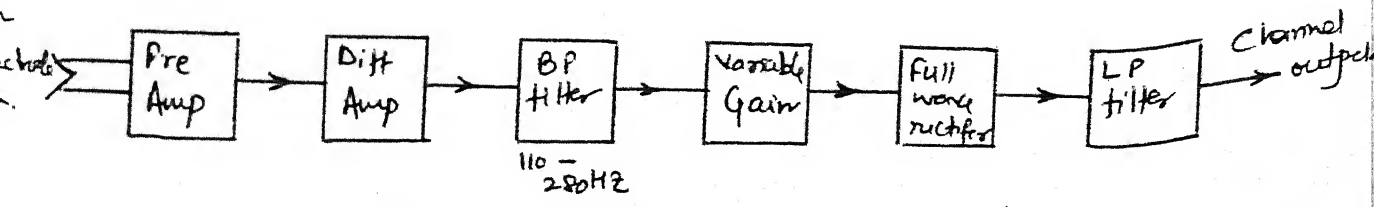


Fig. 18: Block diagram of EMG channel

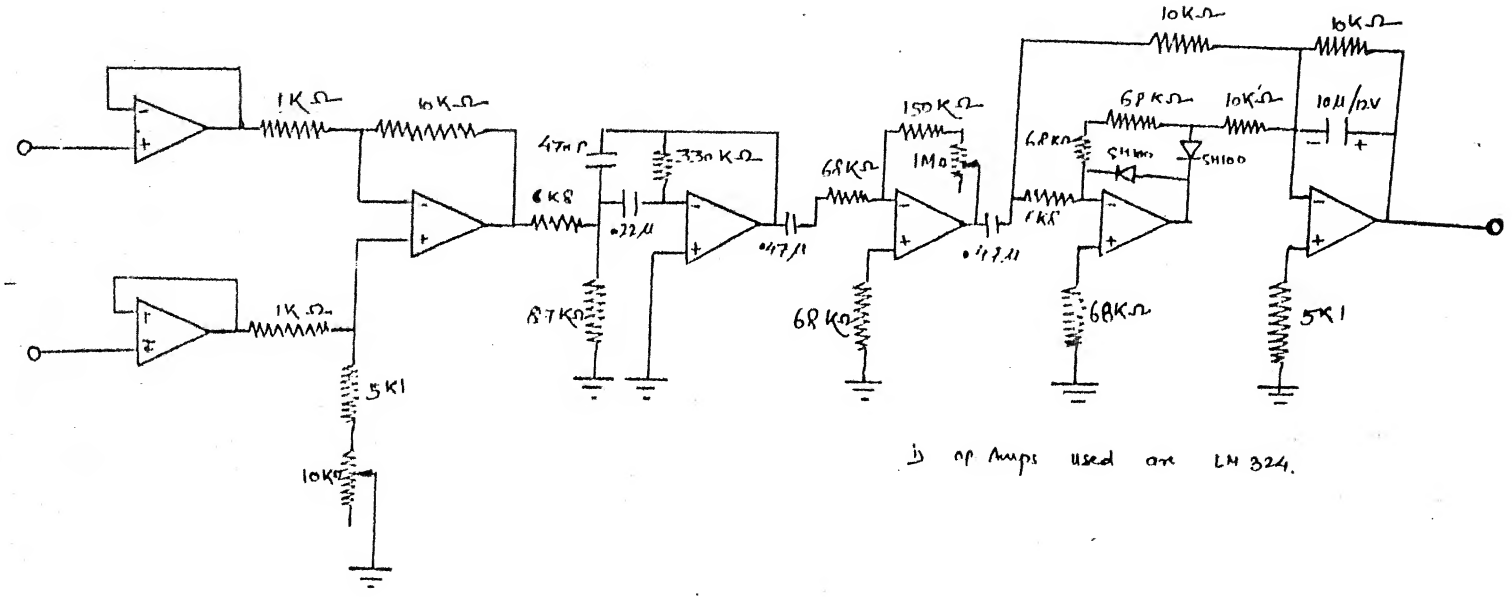


Fig. 19: Circuit diagram of EMG channel

CHAPTER 8

ELECTROMYOGRAPHY - CLINICAL USAGE

8.1 Electromyography in Myopathological Diseases:

The electromyograph recorded carries tremendous information about the state of muscles in the body..

R.D. Fusfeld [6] has shown the use of electromyograph for pathological usage. The pathologic process gets disorientated in case of a myopathic disease because of random disintegration of the muscle. As a result the number of fibres in a motor unit is reduced. This leads to a reduced and fragmented action-potential being generated. In case of severe disorder, the change in the interference pattern of the action-potential may be appreciable on the CRO screen. However an instrument has been developed [6] by Fusfeld which can analyse an electromyograph and can be used for diagnosis. The instrument gives the zero crossing rate and negative wave duration of the electromyographic signal. The variation in the above mentioned rates in a diseased hand is given in Table 1 and Table 2.

8.2 Myoelectric Control of An Artificial Hand:

The perception of a phantom limb prevails even in an amputee. When such a person is asked to carry out phantom movements, specific muscle contraction are produced in the stump. Now if a number of surface electrodes are placed

Table 1. Quantitative electromyographic data in a normal control group of 15 subjects

	Average	Standard deviation	Range
Zero crossing rate	169	30	105-240
Peak rate	240	45	180-350
Negative wave duration, ms	2.56	0.58	1.72-3.99
Wave rise time, ms	0.86	0.13	0.63-1.10

Table 2. Quantitative electromyographic data in patients with muscle disease

Subject	Diagnosis	Zero crossing rate	Peak rate	Negative wave duration	Wave time rise
				ms	ms
1	Dermatomyositis	340	440	0.55	0.52
2	Dermatomyositis	280	560	1.69	0.51
3	Polymyositis	250	360	1.51	0.63
4	Duchenne m.d.	110	220	1.68	0.49
5	Duchenne m.d.	120	195	2.74	0.60
6	Duchenne m.d.	105	120	2.90	0.53

around the stump, myoelectric signals of varying strength will be picked up by the electrodes.

Dr. G.C. Ray etc. did carry out the experiment [7] on a below elbow amputee, based on above line of thinking. Five electrodes were used and six basic movements of FF (finger flexion), FE (finger extension), WF (wrist flexion), WE (wrist extension), P (pronation) and S (supination) were performed. Table 3 gives the voltages obtained from the various electrodes for different movements of a normal subject and Table 4 for a below elbow amputee.

To realise a hardware, electrodes 2, 4 and 5 were selected and the detector levels were set at 0.6, 1.0 and 0.6 V respectively. The block diagram of the scheme is shown in Figure 24. The logic gates will now give output of '1' or '0' depending upon whether the signal level is above or below the detector level. Thus we can have binary states of 111, 101, 011, 100, 001 and 000 for FF, FE, WF, WE, P and S respectively. The output of the discriminator circuit can now be used to control the movement of three motors to perform the above mentioned movements.

A similar approach has been adopted by Almstrom and Herberts [8] for a multifunctional hand prosthesis. The difference in the two approaches is that the latter uses a computer for statistical discrimination of function rather than fixing the detector levels on the best suitable observation.

Electrode pair number	Muscles	E.M.G. potentials					
		FF	FE	WF	WE	P	S
		V	V	V	V	V	V
1	Extensor carpi radialis longus	1.70±0.50	0.32±0.16	0.04±0.02	1.68±0.36	0.14±0.12	0.06±0.03
2	Extensor carpi radialis brevis	1.09±0.37	1.46±0.34	0.04±0.02	2.07±0.38	0.36±0.10	0.08±0.06
3	Flexor carpi radialis	0.34±0.20	0.26±0.20	1.96±0.45	0.08±0.08	1.42±0.58	0.04±0.03
4	Palmaris longus	1.74±0.49	0.20±0.15	1.45±0.31	0.04±0.03	0.35±0.14	0.08±0.05
5	Flexor carpi ulnaris	1.40±0.40	1.16±0.38	1.70±0.23	0.03±0.02	1.0 ±0.22	0.07±0.06

Table 4. Potentials recorded from the stump of the amputee shown in Figure 24

Electrode pair number	Muscles	E.M.G. potentials					
		FF	FE	WF	WE	P	S
		V	V	V	V	V	V
1	Extensor carpi radialis longus	1.6	0.3	0.2	1.2	0.05	0.4
2	Extensor carpi radialis brevis	0.8	0.9	0.2	0.4	0.05	0.9
3	Flexor carpi radialis	0.3	0.2	0.4	0.1	0.4	0.05
4	Palmaris longus	2.3	0.3	0.9	0.1	0.5	0.05
5	Flexor carpi ulnaris	2.0	1.5	0.6	0.2	0.02	0.03

8.3 Conclusion:

Myography has reached much beyond the limits and usage given above. Now even a forequarter amputee can be provided with a myoelectric hand which can be controlled by pulses drawn from the shoulder blades of the amputee. A twitching of the shoulder will make the artificial hand move as desired. Demand pacemakers are another example.

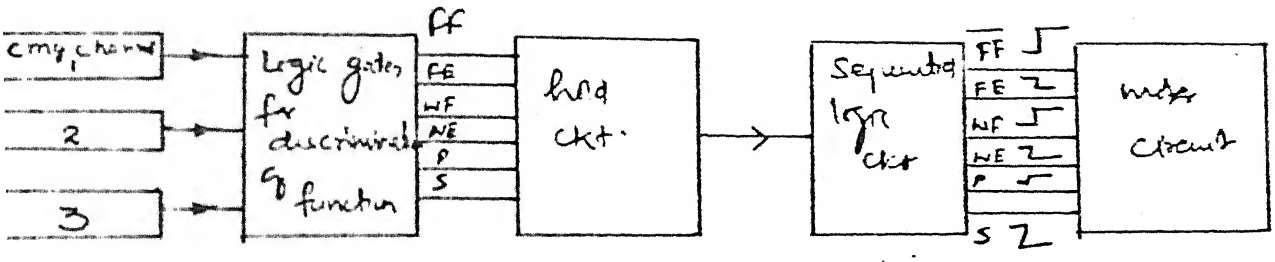


Fig. 24: Block diagram for myoelectric control of artificial hand

CHAPTER 9

EFFECT OF SWEATING ON MYOELECTRIC SOURCE

9.1 Introduction:

In the previous chapter we have seen that prosthesis depends upon discrimination of function depending upon the amplitude of the myoelectric signal solely. In the process the assumption made inherently is that the magnitude of the EMG signal would remain constant for a particular movement independent of time. However this assumption falls way apart from reality especially in a tropical country like India where sweating is a common phenomena and some persons sweat profusely.

The sweat layer formed on the surface of the skin causes a virtual short circuit of the surface electrodes. This causes a variation in the input impedance of the channel and since the sweat layer varies randomly, the variation in the input impedance is random and as a result a random variation in output voltage will be observed for the same movement of the muscle.

Ray and Guha have done extensive work on the effect of sweating on myoelectric source [9]. Figure 25 shows effect of sweating on the surface EMG potential. A three electrode systems was used with a spacing of 2.25cm between active electrodes and the earth electrode placed 3 cm away.

The initial increase in EMG potential is attributed to better contact resistance due to the salineness of the sweat layer. As the sweat layer increases, the high input impedance of the preamplifiers is partially shunted and the EMG channel picks up ~~more~~ less and ~~more~~ less voltage. The decrease in signal amplitude was as large as 13%, 33% and 44% at 38°C, 32°C and 35°C respectively.

9.2 Electrical Modelling of the Sweat Layer :

It would be useful to have an idea of the changes in the electrical circuit due to sweating. Figure 26 shows an approximate model of the effect of sweat layer. The muscle has been assumed to be resistive for simplicity. R_{12V} is the muscle resistant and R_{12S} is the sweat layer resistance.

9.3 A Scheme for Gain Compensation:

It was perceived by the authors that if the gain of the EMG channel itself is increased in a process depending upon the variation in sweat layer, the problem can be countered. Figure 27 shows the block diagram of the scheme suggested by the authors and Figure 28 shows the equivalent circuit taking into consideration the sweat layer. It was also shown by the authors that for linear deposition of sweat layer the fall in output voltage is also linear (Figure 29).

The scheme is based upon injecting a 8 KHz a.c. voltage in the muscle and the amplitude is kept low (order of mv). The amplitude of this high frequency voltage, picked up

by the electrodes across 1 meg-ohm resistors, is shunted by the R_{12} (the resultant sweat and muscle resistance R_{12S} and R_{12V}), R_{23} and R_{31} between the different electrode pairs. These resistances have been measured and in absence of sweating they are as follows - R_{12} = 32 K, $R_{31} = R_{23}$ = 43 K and they fall to 20 K and 31 K respectively.

Assuming a very high input impedance of the preamplifiers, the voltage picked up by the electrode pair 1-2 can be given as

$$V = i \cdot \frac{R_{31} R_{12}}{R_{12} + R_{23} + R_{31}}$$

where i is the current.

The voltage picked up by the electrodes also contains the EMG signal. The 8 KHz signal and EMG signal are band pass filtered respectively. 8 KHz channel voltage is peak detected and then applied to a slope changer (a transistor circuit) Figure 30 which controls the AGC amplifier of the EMG channel.

9.4 Conclusion:

The major drawback in the system are as follows:

(i) The muscle is a volume conductor and its specific resistance may change due to contraction (hardening). This will lead to the 8 KHz channel picking up more voltage. Thus a overcompensated channel at moderate contraction may turn into an undercompensated channel.

(ii) The muscle resistance has been taken to be resistive. However it has got a capacitive component also.

(iii) The characteristic of a transistor slope changer may vary when a transistor is replaced.

(iv) It is an open loop control system and has its inherent drawbacks.

(v) Contact resistance has not been considered anywhere.

(vi) Injection of 8 KHz signal may be objectionable to clinical use.

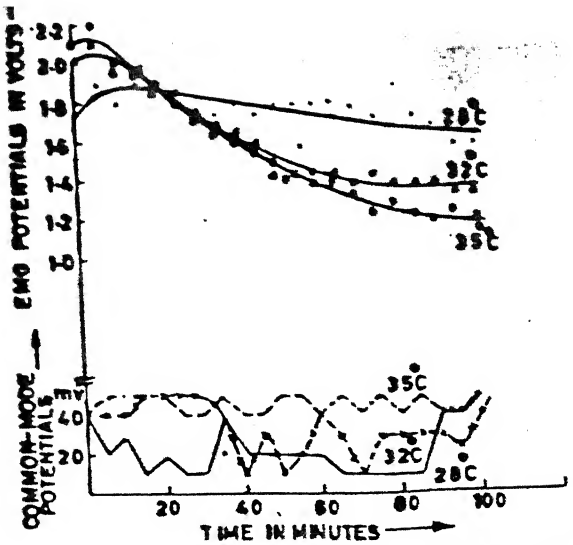


Fig. 25 Reduction in the amplitude of the surface EMG due to the deposition of the sweat layer shunting the input impedance of the preamplifiers.

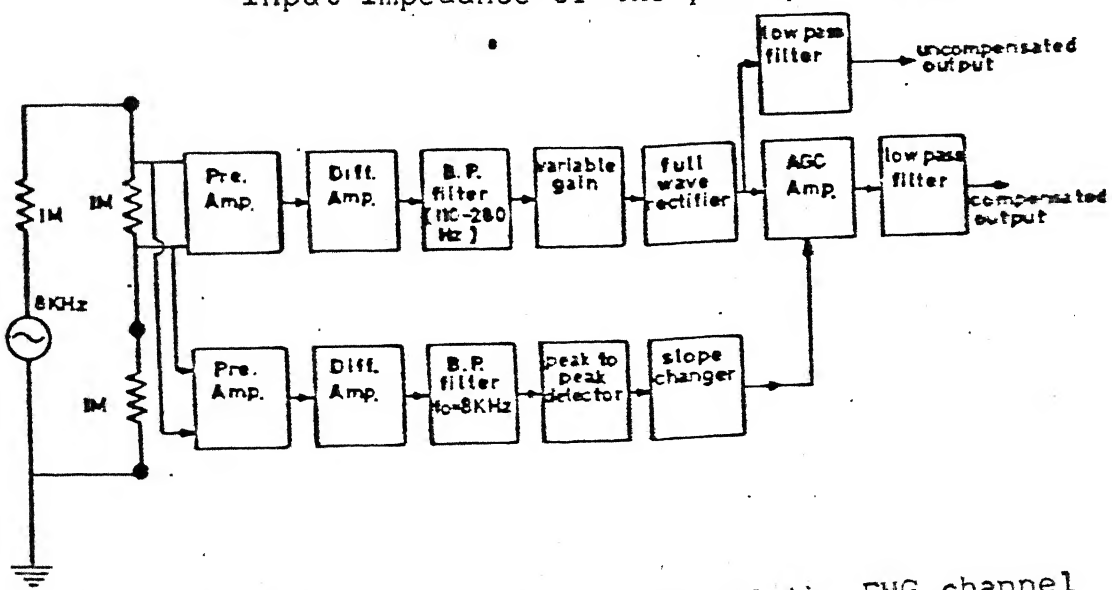
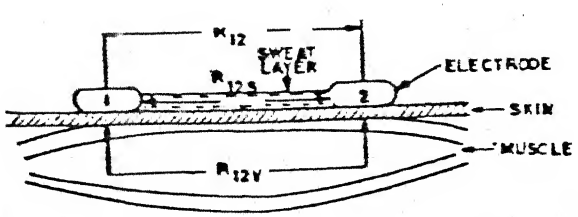


Fig. 27 Scheme for gain compensation of the EMG channel using the voltage drop across the sweat layer.



istance of the sweat layer (R_{12s}) resistance of the volume conductor (R_{12v}) appear in parallel oss any electrode pair.

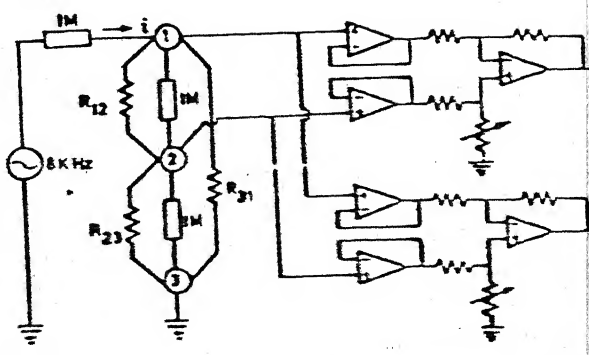


Fig. 28 Equivalent electrical repres of the sweat layer and the v conductor.

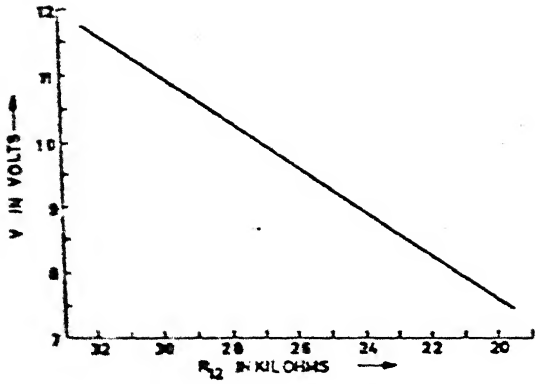


Fig. 29 Fall in voltage due to sweat layer deposition.

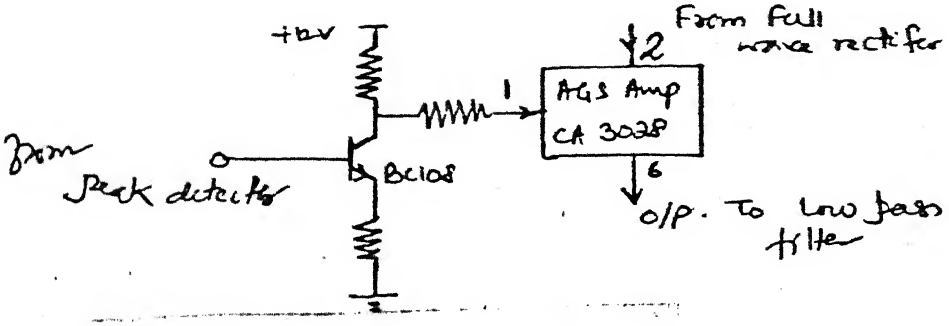


Fig. 30 Slope changer.

CHAPTER 10

SWEAT COMPENSATION OF EMG CHANNEL USING MICROCOMPUTER 8748

10.1 Introduction:

The scheme for gain compensation using a microcomputer 8748 is shown in Figure 31. The basic scheme indicated in [9] has been retained. However the drawbacks of the previous scheme has been taken care of by the following:

(i) By using a closed loop control system, it has been possible to nullify the effects of random deposition of sweat layer which is not necessarily linear.

(ii) Unequal variation in the contact resistance can also be compensated.

(iii) The variation in muscle resistance due to muscle hardening on contraction may be taken care of by keeping a reference voltage of 8 KHz signal recorded under contraction.

10.2 Scheme for Compensation:

The system works as follows. First dry skin condition is maintained and the signal is processed in the 8 KHz channel. After band pass filtering, the 8 KHz signal is peak detected and applied to an A/D converter. This reading of the A/D converter referred to as the "dry skin reference voltage" is stored in the EPROM section of 8748. However provision has

Acc. No. A 38922

also been made to take the first reading of the 8 KHz channel as the reference voltage (assuming a dry skin condition was maintained). Subsequently, during normal operation the 8 KHz signal amplitude (duly effected by sweat layer) is measured by the A/D converter and then compared with the reference stored in the EPROM. Depending upon whether the current reading is smaller or greater than the reference, an incremental output is sent by the microcomputer to a DAC. The output of this DAC than controls the gain of a voltage controlled amplifier in the 8 KHz as well as the EMG channel. The DAC steadies at a value where the current reading equals the reference.

Since it can be assumed that the variation in sweat layer will not be continuous, the programme of the computer may be adjusted to stop the gain compensation for a specified amount of time during which the 8 KHz injection can be stopped. This stopping of the 8 KHz signal injection into the body makes the systems available for diagnostic purposes.

The circuit description is given in Figure 32. The working of the various parts of the circuitry is explained below.

10.3 8 KHz Oscillator:

It is a Wien-Bridge oscillator. The circuit is given in Figure 33. R_1, C_1, R_2 and C_2 form the Wien-Bridge. The diodes in the feedback loop provide amplitude stability by controlling the feedback. If $R_1 = R_2$ and $C_1 = C_2$ then

$$f_o = \frac{1}{2 R_1 C_1}$$

The potentiometer P_1 is adjusted to obtain the oscillations. The frequency stability depends upon R and C and is good. The amplitude of oscillation depends on P_1.

For this project R_1 = 2 K and C_1 = 0.01 Mfd gives a frequency of 7.96 KHz.

Since the operation of the whole system depends upon the 8 KHz channel it is very important to ensure that the 8 KHz oscillations are produced continuously (which may vanish in case the potentiometer P_1 gets disturbed). To give the indication of such a case an indicator circuit has been appended (Figure 34). Also the amplitude of oscillations must remain constant (else the reference will loose its validity). To achieve this two 4.3 V zeners have been connected back to back giving approximately a 10 V output. This takes care of any variation in the amplitude. However a little distortion is induced, but its effect is not low enough to effect the EMG channel.

10.4 8 KHz Band Pass Filter:

A multiple feedback band pass filter has been used (Figure 19) where $C_3 = C_4 = C$. For this project the filter components were chosen as R_1 = 68 K, R_2 = 3.9 K, R_3 = 1.15 meg-ohm and C = 330 PF. This gives a gain of about 10 and a centre frequency of about 7.95 KHz. The BW is very narrow ($0.1\ f_o$).

10.5 The Peak Detector:

Since the objective was only to get a d.c. voltage proportional to the a.c. input, a diode peak detector has been used (instead of a precision peak detector). This reduces the cost and does not effect the efficiency since two voltages are merely compared and hence absolute value is not important.

The circuit is drawn in Figure 35. The operation is like this. During negative half cycle, diode D_1 conducts and C_1 charges with the polarity shown. When potential at A is large enough diode D_2 also conducts and capacitors C_1 and C_2 share the charge. Capacitor $C_1 >> C_2$ and hence most of the voltage appears across C_2 (as charge is shared equally). During the half cycle only D_2 conducts and C_2 charges via D_2 and thus a voltage addition takes place.

Thus the output is sum of the two peaks minus the diode drops. An a.c. to RMS converting chip like AD536 could also have been used. But the increase in performance is not worth the increase in cost.

For this project C_1 = 0.22 Mfd, C_2 = 0.022 Mfd, R = 1 meg-ohm. Figure 36 shows the characteristic.

10.6 Voltage Controlled Amplifier:

Field effect transistor BFW11 has been used as a voltage controlled resistor. The circuit is shown in Figure 37. As the control voltage at the gate is varied from 0 to -3 volts, a gain of 0.75 to 3 was obtained and this range is sufficient to take care of the variation in output caused by

a deposition of sweat layer between the electrodes.

The characteristic is plotted in Figure 38. Even though the behaviour is non-linear (due to inherent drawback of a FET) it does not really pose any problem because of the closed loop nature of the control loop.

10.7 A/D Converter AD7574:

The primary features of AD7574 are given at Appendix A. It has been used in the unipolar mode (as the output of peak detector will always be positive). AD7574 gives magnitude output. The reference voltage used for this project was -12 V (obtained by a LM320 voltage regulator chip) for ease of availability (even though recommended voltage is -10 V). Therefore one count of AD7574 will now be equal to $\frac{12}{256}$ V or 47 mv approximately.

10.8 D/A Converter AD558:

The data sheet for AD558 is given at Appendix B. For this project it was used in the 0 to 2.56 V range (as the FET control voltage range is upto -3 V). Negative voltage for the gate control of the FET was obtained by using 741 opamp as an inverter. One count of the DAC will now correspond to 10 mv which gives a fantastic incremental voltage for the FET. This was the reason it was preferred to use AD558 and an inverter rather than using a bipolar DAC (higher output range thus larger incremental step and hence not suitable).

```
        OUTL P2,A;
        ENTØ CLK;          Output 2MHz clock at TØ pin.

LOOP1:  IN A,P2;           Wait for "RUN" signal.
        JBØ LOOP1;
        JB1 NORML;

TEMP:   MOV A1 # 5AH;      Voltage controlled amplifier
                           gives unity gain for a control
                           voltage of (-0.7 V), the corres-
                           ponding DAC word is 5A.
        MOV RØ, # 21H;
        MOV @RØ,A;         Save DAC word at RAM location 21.
        OUTL P1,A;         5A to DAC
        CALL HOLD;         Give delay for the circuit to
                           stabilise
        CALL ADC;          Read A/D converter, reading
                           left in accumulator.

GAIN:   MOV RØ, # 2ØH;
        MOV @RØ,A;         Reference voltage stored at RAM
                           location 2Ø.

ADJST:  CALL HOLD;         Give delay
        CALL ADC;          Read ADC
        CPC A;
        ADD A, # Ø1H;      Take 2's complement of current
                           reading.
        MOV RØ, # 2ØH;     Memory pointer (RAM) to 2Ø (i.e.
                           reference voltage).
        ADD A, @ RØ;       Add reference voltage to current
                           reading.
        JZ WAIT;           Reference voltage = current voltage.
        JNC DECR;          Reference   current, so decrease
                           the DAC word

INCR:   MOV RØ, # 21H;     Old DAC word to A
        MOV A, @ RØ;
        INC A;             Ref   current reading; so
                           increase the DAC word.
        JZ MAX;            DAC word = ØØ of incremented at FF.

NEXT:   OUTL P1,A;         New DAC word to DAC
        MOV RØ # 21H;
        MOV @ RØ,A;        Some new DAC word
        JHP ADJST;         Repeat the process

MAX:    MOV A, # ØFFH;     Set maximum DAC word and
        JMP NEXT;          Send to DAC

DECR:   MOV RØ, # 21H;     Old DAC word to A
        MOV A, @ RØ;
        JZ NEXT;           DAC word is minimum
        DEC A;
        JMP NEXT;
```

```
WAIT:   MOV A,# ØEFH:       Disconnect oscillator by sending
        OUTL P2,A;          'Ø' to 5537 control pin.
        MOV RØ,# ØØH;       Initialise delay
        MOV R1,# ØØH;       resistors.
        MOV R2,# ØØH;
        MOV R3,# 5ØH;

DRØ:    INC RØ;             Give delay of
        MOV A,RØ;           aproximately
        JZ DR1;
        JMP DRØ;

DR1:    INC R1;             8.389 x 80 / 60 = 11.18 minutes
        MOV A,R*;
        JZ DR2;
        JMP DRØ;

DR2:    INC R2;
        MOV A,R2;
        JZ DR3;
        JMP DRØ;

DR3:    DEC R3;
        MOV A,R3;
        JZ END;             Delay completed
        JMP DRØ;

END:    MOV A,# ØFFH;
        OUTL P2,A;          Send '1' to 5537 control pin
        JMP ADJST;          Do gain adjustment

NORML:  MOV A,# 5AH;
        OUTL P1,A;          5A to DAC
        MOV RØ# 21H;
        MOV @ RØ,A;         Same DAC word
        MOV A,# ØØH;        Reference voltage
        MOVP3 A, @ A;       From location 3ØØ to accumulator.
        JMP GAIN;           Do gain adjustment.
```

Subroutine ADC: This subroutine reads the contents of the A/D converter and leaves the reading in the accumulator. It is located at location 1AØ of the PROM section.

```
        ORG 1AØH;
ADC:    MOV A,# ØFBH;       Send 'start' convertion signal
        OUTL P2,A;          to AD7574, bit B2 made low.

LOOP2:  IN A,P2;            Wait for Busy pin of
        JB3 READ;           AD7574 to go high
```

```
READ:   INS A,BUS;          Conversion complete, read data.
        MOV RØ,A;
        MOV A,# ØFFH;       Remove start conversion signal.
        OUTL P2,A;
        MOV A,RØ;
        RETR
```

Subroutine HOLD: This subroutine develops delay after a new word has been sent to the DAC. This delay is required to take care of the time constant of the peak detector. This subroutine is located at location 1DØ of the PROM section of 8748.

```
HOLD:   ORG 1DØH;
        MOV RØ,# ØØH;       Initialise
        MOV R1,# ØØH;
        MOV R2,# 2ØH;       the delay resistors.
DIRØ:   INC RØ;             Start delay
        MOV A, RØ;
        JZ DIR1;            period.
        JMP DIRØ;
DIR1:   INC R1;
        MOV A,R1;
        JZ DIR2;
        JMP DIRØ;
DIR2:   DEC R2;
        MOV A,R2;
        JZ AHEAD;           Delay completed
        JMP DIRØ;
AHEAD:  RETR
```

10.10 PCB Design:

The PCB was designed with special emphasis on the size of the whole unit. As a result the PCB looks a bit crowded. The layout is given in Figure 40. Working model has been fabricated based on the above design.

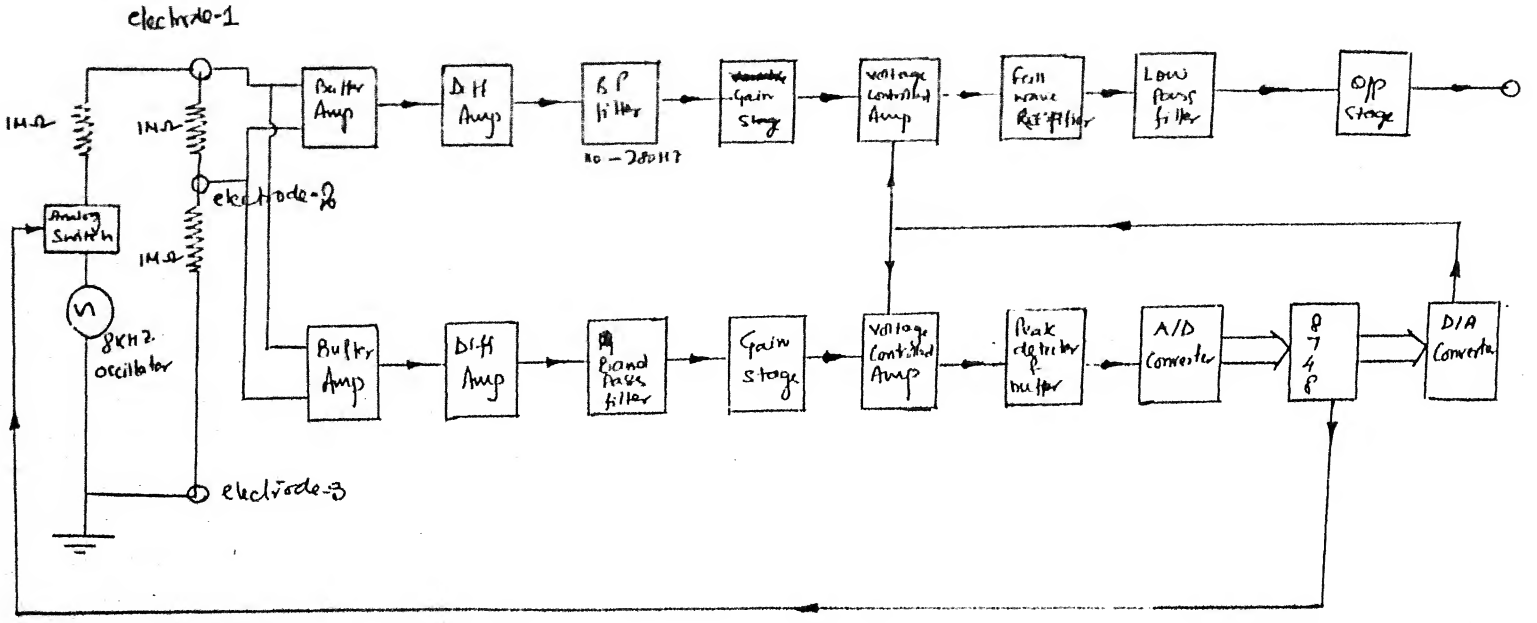


Fig. 31: Block diagram of digitally controlled sweat compensated EMG channel

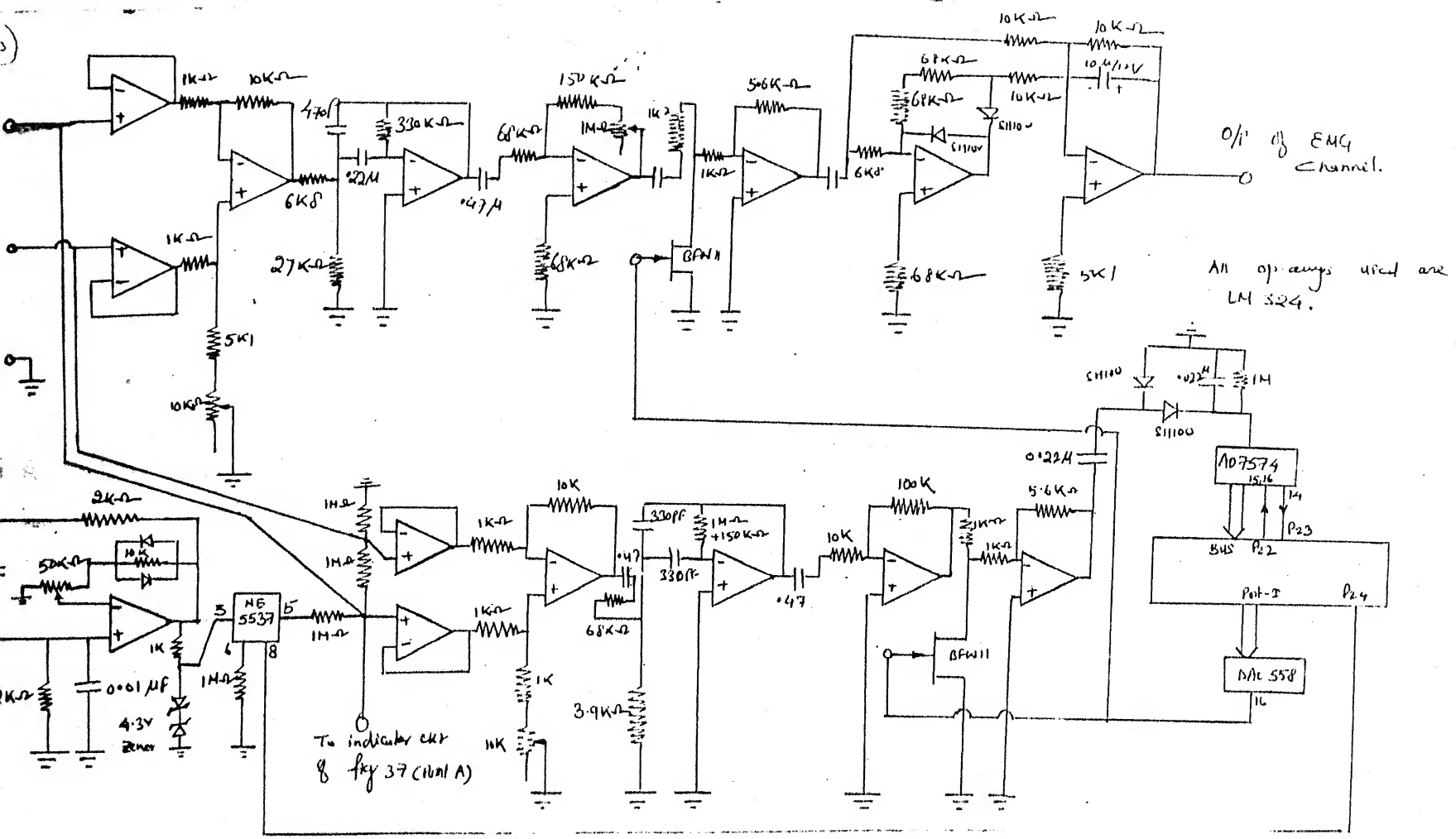


Fig. 32: Circuit diagram – use of digital computer for sweat compensation

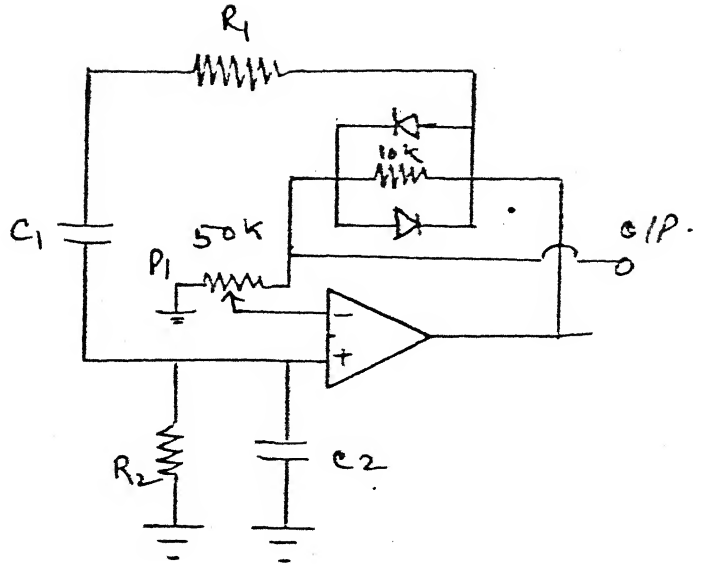
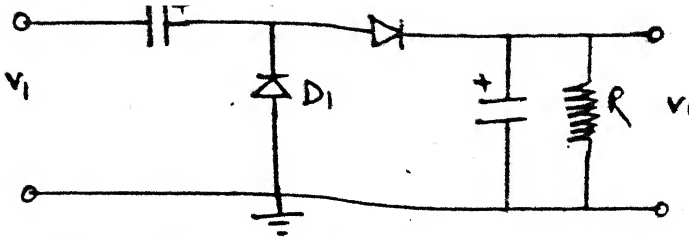


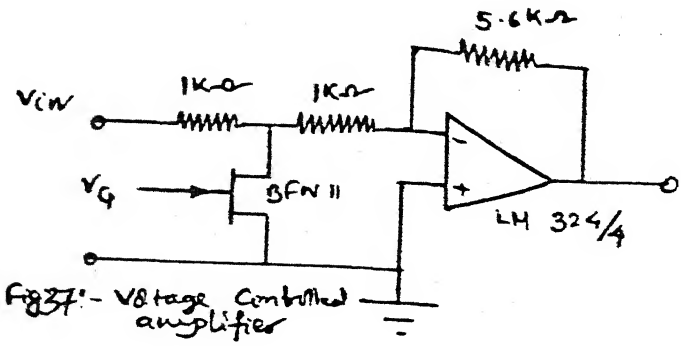
Fig. 33: Wiew Bridge Sine Wave Generator



$RC2 \gg \frac{1}{f}$

$C_1 \gg C_2$

Fig 35:- Peak detector



$0 < V_G < -3$ volts.

Fig37:- Voltage Controlled amplifier

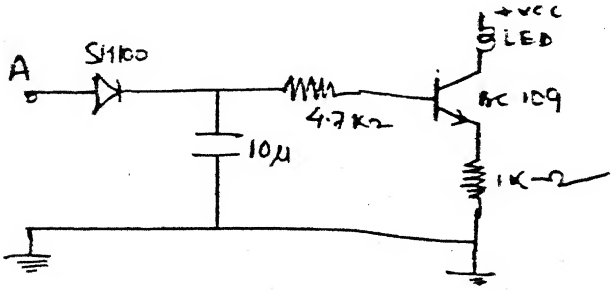
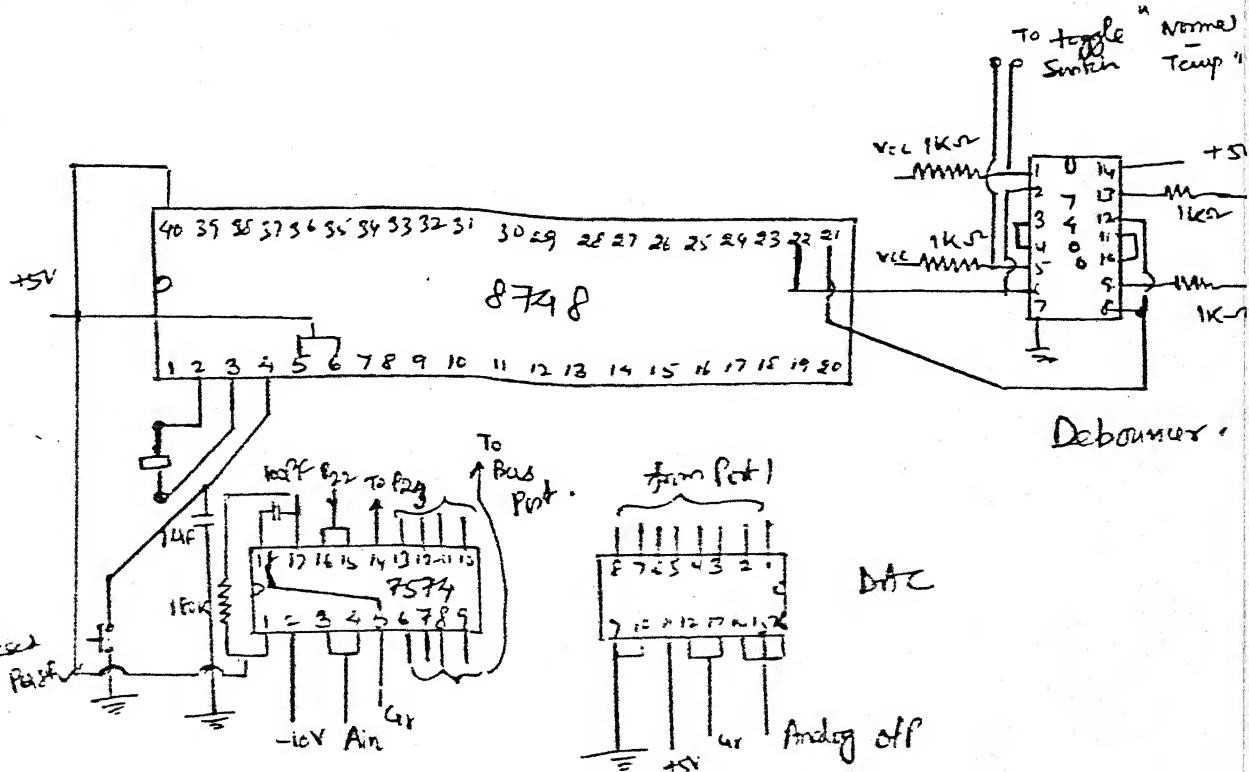
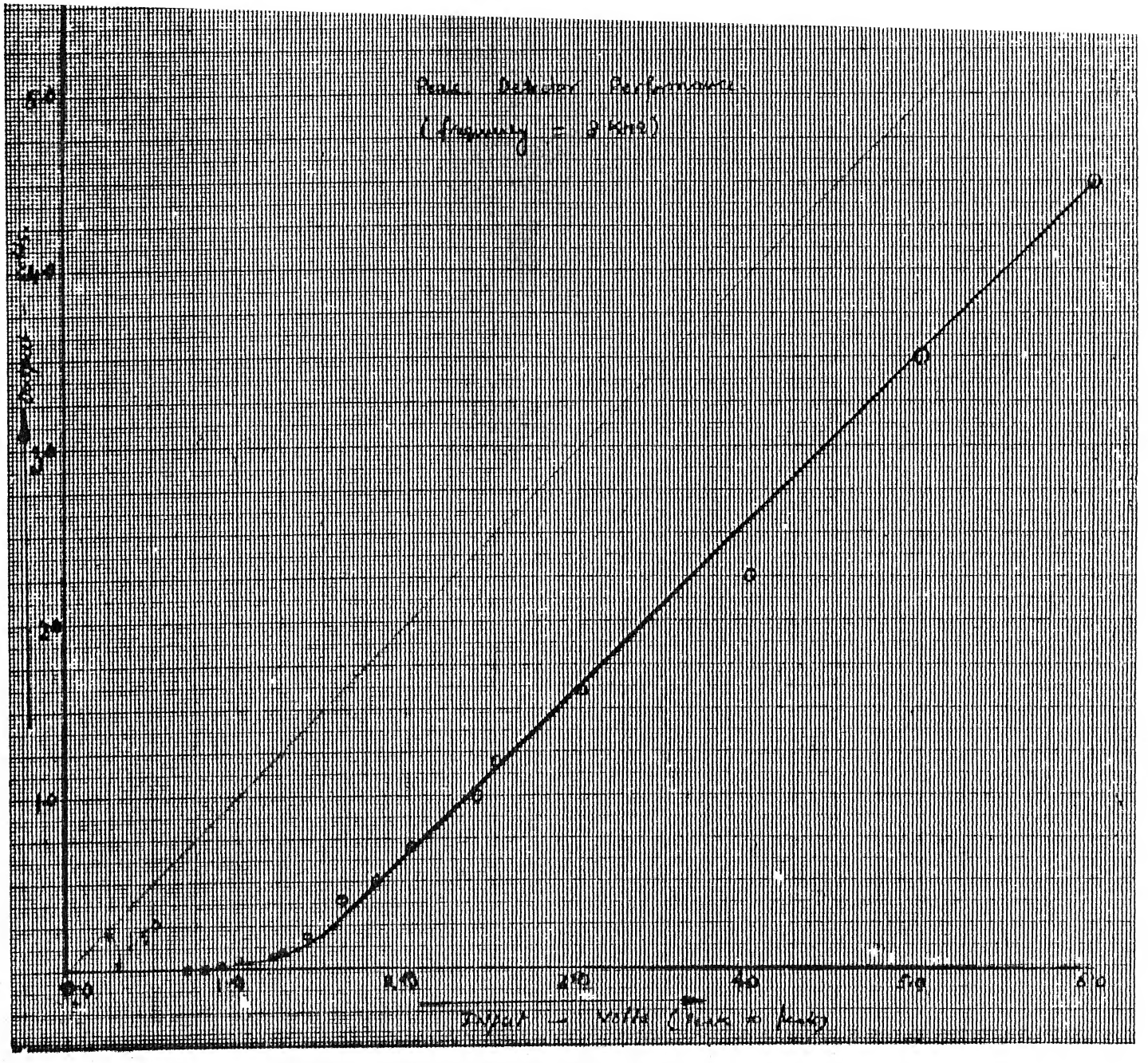


Fig. 34: Indicator Circuit





...characteristics

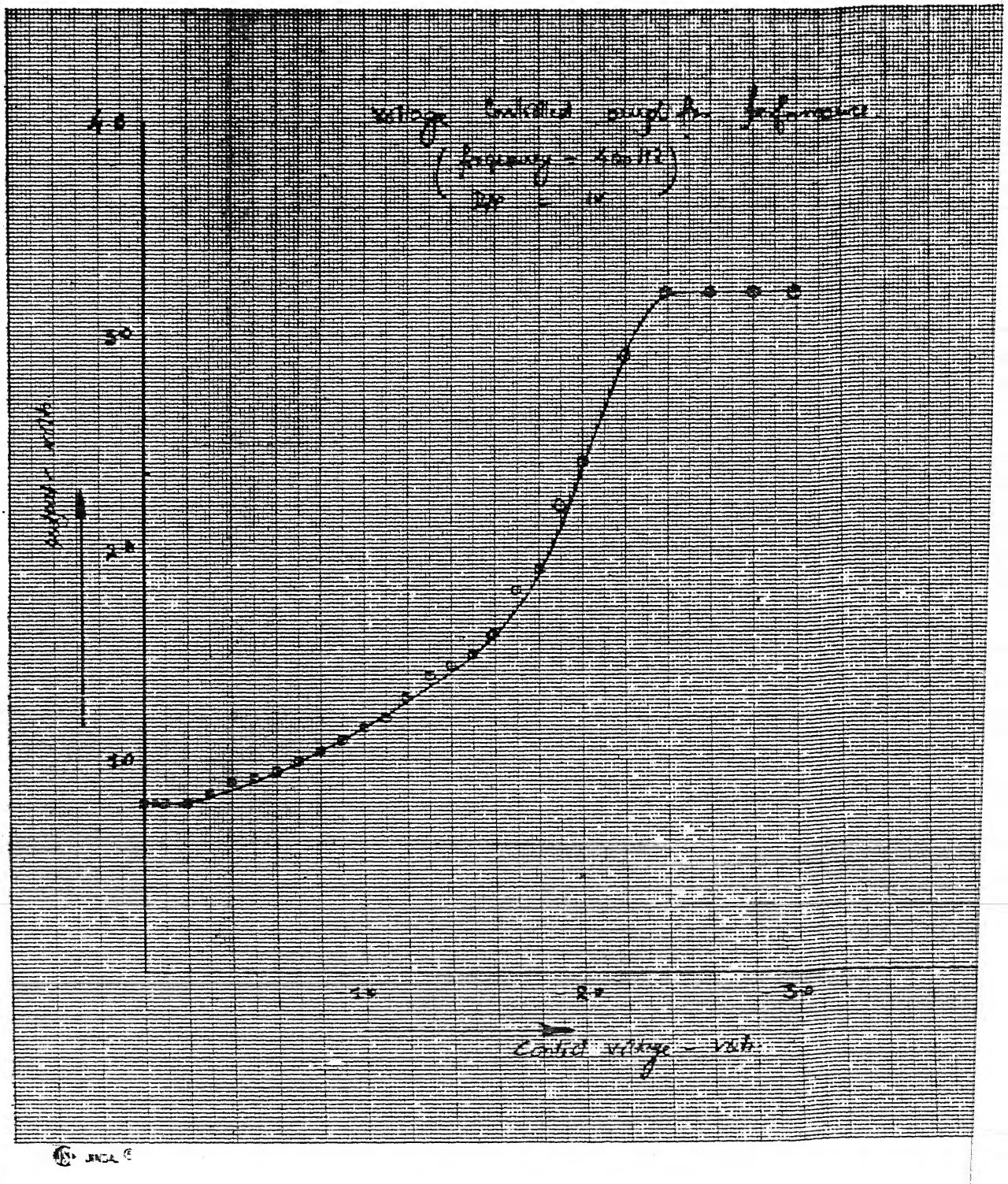
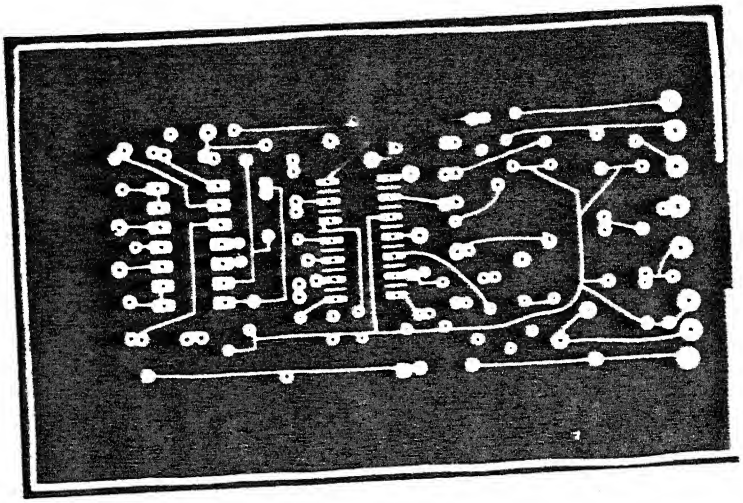
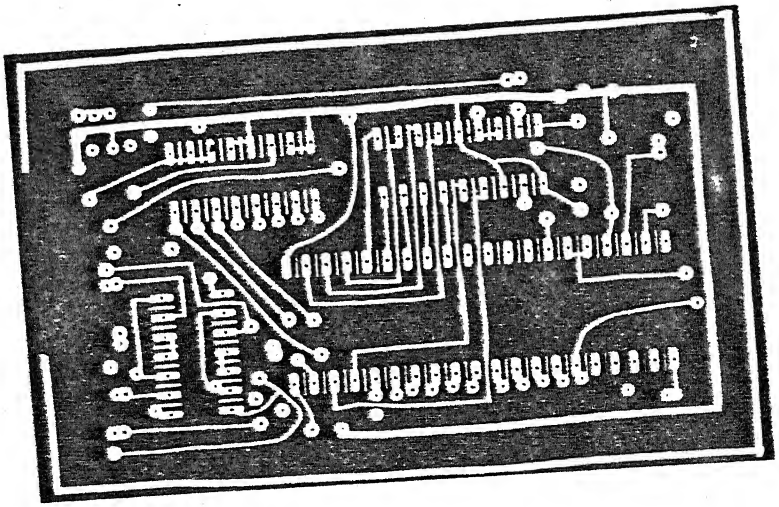
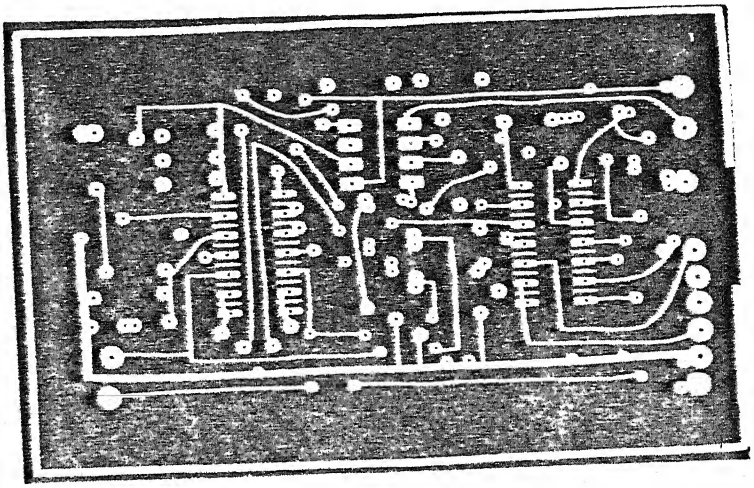


Fig. 38: Voltage Controlled Amplifier Characteristics







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